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LEUKEMIA, POLYCYTHEMIA AND RELATED DISEASES*

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AS WITH all study, of disease or any pursuit of learning, it is necessary to pause occasionally to take stock of one's present position. We of The Ohio State University College of Medicine are privileged in your permitting us to share this inventory period with you. The strides that have been made in the therapeutic management of certain phases of the hematologic dyscrasias during the past decade, some of which we are discussing this morning, have been somewhat amazing even to the most conservative. We feel that we are in a period of optimistic transition and that we are entering the second half of this twentieth century with an enthusiasm and optimisim which have not been equaled since the turn of the century when the ideas of Ehrlich, Röntgen, the Curies and other pioneers were being translated into practical application toward the alleviation of man's illnesses. The analogues of folic acid, urethane, nitrogen mustard, selective radiation with artificially radioactive isotopes—all of these materials have accomplished, in certain instances, therapeutic effectiveness which has been most encouraging. You have heard over the past several days others discuss phases of these progresses. This optimism is not false for we are fully aware of the magnitude of the unknown problems that are presented. The surface has only been scratched, but a scratch has been made and for this we are grateful.

The hematologic system is a definite, integrated, physiologic system in the body the same as the gastrointestinal, respiratory, reproductive and other systems. Certain elements of the reticulo-endothelial system in the bone marrow, lymph nodes, liver and spleen are concerned with the production of the formed elements of the blood —the red blood cells, the white blood cells and the platelets. Other elements of the reticulo-endothelial system, the clasmatocytes (macrophages), located principally in the liver, lymph nodes, spleen and lungs, are concerned with the removal of the aged, senile and debilitated cellular formed elements. The resulting picture that we see in the peripheral circulation is a balance be-

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tween these two mechanisms, namely, the hematologic equilibrium. When there is a significant quantitative disruption of this equilibrium, disease is manifested clinically. This morning my discussion will be limited to those diseases where the hemopoietic mechanisms go "beyond the call of duty," producing an unbridled proliferation of the formed elements of the blood. The causes for this lack of "crop control" are about as vague to us at the present as our understanding of the lack of control in our economic order. Polycythemia vera, the syndrome in which there is a *triune hyperplasia* of all the hematologic formed elements originating in the bone marrow, will be discussed initially. Then, secondly, a résumé on our current thinking of the leukemias—myelogenous, lymphatic, and monocytic, both acute and chronic types—will be made.

POLYCYTHEMIA VERA

In the earlier descriptions of polycythemia vera, it was not recognized that there was a *triune* hyperplasia; only the red cell elevations were noted as is indicated by the misnomer, polycythemia *rubra* vera given the disease. It is now recognized as a pan-marrow hyperplasia involving the granulocytes and platelets as well as the red blood cells. The reason for this unrestrained production is unknown. None of the numerous hypotheses that have been offered is entirely satisfactory. There does seem to be associated in a number of instances a familial tendency towards its development. The symptoms may be extremely protean, ranging from a vague increased fatigability to frank manic-depressive psychotic states. Therapy, in the past, has been focused mainly toward diminution of the erythroid element with agents such as phenylhydrazine, total body roentgen irradiation or repeated venesections with little or no attention being given the other elements. In 1941 the radioactive isotope of phosphorus (P^{32}) was first made available to our laboratory and after nine years' experience with this agent we feel that it is the treatment of choice for this disease entity.

Administered phosphorus, whether radioactive or non-radioactive, has a predilection for deposition in bones and the nuclei of rapidly multiplying cells. Only beta radiations, whose penetration of tissue is limited to approximately 2 centimeters, are emitted. The half-life of 14.3 days is sufficiently short for allowing careful dosage regulation using the peripheral hematologic picture as the guide. Dosage depends on the individual sensitivity of the marrow cells. This varies greatly from individual to individual and extreme caution is necessary for proper regulation. Inasmuch as we are dealing with three formed elements with varying developmental life spans—the red cell 120 days, the granulocytes approximately fourteen days from the blast to the polymorphonuclear forms, and the platelets four to five days—it is necessary to keep all of these time elements in mind when administering radioactive phosphorus. It is a two-edged sword and serious complications can result if care is not taken. Frequently repeated complete hematologic studies are an absolute necessity for proper control of this means of internal irradiation.

In the initial control of a new case, venesections are freely utilized in order to give immediate symptomatic relief, since the maximum effect on the red cells per se by the initial phosphorus therapy will not be reflected in the peripheral blood before forty to sixty days. With the shorter developmental spans in the platelets and the granulocytes the response on these elements is seen much earlier. In the average case 3 millicuries of radioactive phosphorus (P^{32}) in the form of sodium dihydrogen phosphate is given orally and observations of the hematologic response over the following two to three weeks are made before additional therapy is given. It cannot be overemphasized that *therapy must be individualized to each patient.* Several reports of the use of nitrogen mustard in the treatment of polycythemia vera have recently ap-

peared. Further evaluation of this extremely potent cellular inhibitor is necessary.

As long as the triune hyperplasia is kept under adequate control, the patient is asymptomatic. The secret of effective therapy is to keep in mind the potential complications; thus being forewarned, they can be prevented. Strangely enough, the two most frequent complications of the disease itself are the two extremes of the coagulation phenomenon: first, intravascular thrombosis and second, hemorrhage. Any of the vessels of the body may be involved in thrombosis—cerebral, mesenteric, pulmonary or peripheral. The increased viscosity due primarily to the elevated erythrocytes but enhanced by the elevated platelets precludes spontaneously occurring intravascular thrombosis. The control of the increased platelet production is of equal importance to the control of the red cells. Kety and his associates have recently shown that the cerebral circulation is extremely slow in polycythemia vera. This so-called "sluggishness" is present in the entire circulation. The impeded hepatic circulation is manifested by low prothrombin and fibrinogen levels. This hypoprothrombinemia is occasionally the cause of serious hemorrhage, such as is sometimes seen when an intracranial vessel is involved. With correction of the increased viscosity, the prothrombin returns to normal levels if the vitamin K supply is adequate. The depressed fibrinogen content of the plasma is reflected in a marked diminution in the sedimentation rate. This, along with the peripheral elevation of the red cells, granulocytes and platelets, reflecting the marrow triune hyperplasia, is an aid in differentiating polycythemia vera from secondary polycythemia. This differential diagnosis is extremely important for therapeutic reasons. In the vera states, inhibitory therapy is indicated; in the secondary polycythemias, treatment directed toward the lowering of the compensatory erythroid hyperplasia per se is contraindicated. Attention is focused on the primary cause of the marrow hypoxia.

The relationship of polycythemia vera to leukemia is not at all clear. Occasional instances are seen which terminate as myeloid leukemia and the number of cases now being recognized and reported are increasing. Whether this metamorphosis is part of the natural course of the disease or is the result of therapy is not known.

In reiteration, radioactive phosphorus, when used with venesections as indicated, has proved the most effective means of controlling the triune hyperplasia of polycythemia vera that we have observed. The frequently debilitating and dangerous complications have been minimized.

THE LEUKEMIAS

Leukemia is not necessarily always fatal. Victims may live long enough to die of some other disease. Some physicians and most patients are not aware of this fact. The trend to more effective therapy in the leukemic states has been quite encouraging. The cure is still far from our grasp, the only present "cure" being a change of diagnosis; however, some success has been attained in lessening pain and debilitation and prolonging life in certain types. This is particularly true in certain of the acute leukemias. In the chronic leukemias—myelogenous, lymphatic and monocytic—roentgen irradiation is still the inhibitory sheet anchor. Radioactive phosphorus (P^{32}) has proved to be a valuable adjunct in those individuals hypersensitive or resistant to roentgen radiation. Chemotherapeutic agents, such as urethane in the chronic myelogenous leukemias and the plasma cell leukemias, and nitrogen mustard in the monocytic leukemias, provide other weapons in our therapeutic armamentarium. Urethane allows for a maintenance inhibitory therapy with daily administration. In chronic myeloid leukemia it has been as successful as roentgen irradiation, in our experience.

According to the polyphyletic theory of blood cell formation the myeloblast, the lymphoblast and the monoblast are cells

derived from the parent reticulum cell. Each of these three blast-type cells gives rise to its respective specific strain of leukocytes. Experimentally it has been shown that certain dietary substances increase maturation and multiplication of each specific cell strain. Nucleoproteins stimulate myelopoiesis; foreign proteins, lymphopoiesis; and lipids, monocytopoiesis. Knowing these facts, we advise elimination of these specific dietary elements in the respective types of chronic leukemias as an addition to the depressant and destructive physical or chemical therapy. It is, of course, difficult to analyze these results statistically; however, nineteen years of experience with the utilization of these dietary limitations in our clinic has given the impression that they are of definite benefit.

In the therapy of the acute leukemias the progress over the past two years has far exceeded that of the preceding past half century. Up to the last several years, therapy in any case of acute leukemia was generally undertaken with trepidation since much of the therapy instituted aggravated the disease process in as many instances as benefit was obtained. With the discovery of the folic acid molecule, chemists began experimenting with minor variations in the molecule, and a series of chemical substances were produced which, though analogues of folic acid, proved to be actually folic acid antagonists. One of these, aminopterin (pterylglutamic acid), has been found to be extremely effective in inhibiting the neoplastic cells of acute lymphatic leukemia. In this specific entity, remissions of six months or longer can be induced in approximately 60 per cent of the cases. These remissions are, for the most part, a return to near normal or normal peripheral hematologic and bone marrow pictures with excellent clinical improvement. The patients are able to return to their normal activities for varying periods of time. Some of these cases have gone for over a year and a half in good remission. This is in marked contrast to the short, ineffective and rather uncomfortable clinical states that were effected in the "remissions" with previous therapeutic agents. Aminopterin has been less effective in controlling the acute monocytic leukemias; however, nitrogen mustard has shown definite benefit in certain instances of the acute monocytic type. Urethane (ethyl carbamate) has been frequently successful in controlling the subacute myelogenous states as well as the chronic myelogenous leukemias.*

And just what can be concluded from these clinical observations? We feel that this is the first time that we have seen any evidence of one specific molecule inhibiting one specific strain of leukemic cells more than another. We feel that this is a period of transition, a transition from the therapeutic era of nonspecific cellular inhibition to one of specific cellular inhibition with specific molecules. It is true that there is much to be desired. No cure has yet been effected. However, remissions have been induced in the past two years, the like of which we have never previously observed with any of the "nonspecific" inhibitory agents. And in this period of transition for the most effective therapy in the acute leukemias specific cellular diagnosis is imperative. Previously, the identification of the specific cell strain involved in acute leukemia was mainly a matter of academic interest; however, now, in order that the most beneficial therapeutic means can be used, specific cellular diagnosis becomes of paramount importance.

The horizon is brighter. We as hematologists have been extremely encouraged with the accelerated progress of the last few years and it is with this encouragement that we are realistically looking to the next few years with optimistic anticipation.

Dr. Hoster, Dr. Morton and Dr. Zanes will consider in more detail specificities of roentgen radiation in both diagnosis and

* Since this presentation the adrenocorticotropic hormone (ACTH) has been widely used for treating all types of acute leukemia. Temporary remissions may be produced in approximately 30 per cent of these cases. It is still only an investigative tool, and not a permanently corrective agent.

therapy of certain of these and other hematologic dyscrasias.

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DISCUSSION

Dr. William E. Howes, Brooklyn, N. Y. Dr. Wright's so-called "specific" therapy for the treatment of polycythemia vera and the leukemias is indeed a novel and rather ingenious approach to the treatment of these various blood dyscrasias. In support of his theory he has summarized the development of the various blood cells from their stem cells, and he has included a time table for the maturation of the various corpuscular elements to be found in the circulating blood, along with a glimpse into that delicate balance maintained between the development of the blood elements as opposed to their destruction.

That radiation therapy be not used in the treatment of secondary polycythemia, he has justly emphasized the characteristic "triune" hyperplasia of the formed blood elements in polycythemia vera. In this regard the use of phenylhydrazine along with total body roentgen irradiation as well as repeated venesection is actually directed toward reducing the red blood cells only. Changes produced in the bone marrow by small doses of radiation are sufficient to startle and confuse the examining hematologist unless he is so apprised. Total body roentgen irradiation is all inclusive and dampens the hematopoietic system in its entirety and is, therefore, in my opinion dangerous. I recall one instance, where, following total body irradiation one polycythemic patient's blood count dropped with alarming rapidity. The picture terminated as that of a myeloid leukemia complicated by an extreme anemia.

Up to the present I have treated with radiophosphorus only those polycythemic individuals who have previously received repeated roentgen treatments and who were considered as roentgen resistant. In each instance a single dose of P^{32} resulted in dramatic relief of the presenting symptoms of headache and dizziness along with a temporary drop in the red and white blood cell count. The dietary suggestions as well as to precede the isotope therapy by venesection are well taken.

Dr. Wright properly states that in the chronic leukemias "roentgen therapy is still the sheet anchor." Again radiophosphorus is a valuable adjunct and is indicated in those hypersensitive or resistant to roentgen irradiation. Here urethane in Dr. Wright's experience has proved as successful as roentgen therapy. Obviously, Dr. Wright has noted no untoward reaction to urethane treatment as he fails to mention possible toxic effect. I therefore add a word of caution, referring to the literature, where 4 cases have been reported with severe reaction, 2 of whom died following urethane in accepted doses. The unrestricted recommendation of urethane therapy should be further delayed until careful trial has been conducted in institutions and under conditions similar to that now being carried out at Ohio State University.

Successful trial of the so-called folic acid antagonists, more particularly aminopterin, represents the result of practical application of brilliant deductive reasoning. Temporary remissions in well over 50 per cent of children so treated have been reported. Each of us should welcome the advent of this new agent and hope that this will lead the way to the discovery of an even more efficacious chemical. You have in all probability experienced that same feeling of hopelessness and frustration when called upon to treat these tragically ill young people.

Dr. Wright offers nitrogen mustard in treatment of acute monocytic leukemia, which has failed to respond to aminopterin. Here, again, a warning note is added: This drug is exceedingly toxic in that it exerts a powerful dampening action upon the entire hematopoietic system, and, in addition, there is serious danger of permanent testicular atrophy. Should there be the slightest possibility of survival this latter complication must be taken into consideration before instituting this therapy. It is to be hoped that in the near future a less toxic mustard will be developed than the bis compound.

Having added these few words of caution, I wish to congratulate Dr. Wright on his informative and instructive presentation of the changing modalities now coming into vogue in the more successful treatment of polycythemia, the leukemias and allied diseases.

Dr. Henry J. Ullmann, Santa Barbara, California. There are several points that should be remembered by those who have not had experience in radiation therapy. Those who have had extensive experience will not need this discussion.

Dr. Howes spoke of injury produced by radiation therapy. To a radiation therapist, injury produced in treating any one of these diseases by radiation means gross overdosage, and a lack of knowledge of what the therapist is doing. The danger in the use of urethane is real, but may be considered a calculated risk. It is still to be evaluated, and I suggest no criticism of its proper use when asking Dr. Wright if the total length of useful life is as good or better under urethane therapy, as by conservative radiation therapy, of the lymphoid leukemias or arsenic in addition to irradiation in the myeloid forms, or by total body irradiation for polycythemia, if the same length of time after treatment is allowed to elapse before the effect is expected. I speak particularly of polycythemia, because the average physician, be he a general practitioner or an internist, is disappointed if he does not find a drop in the cell count in two to three weeks, but he does not expect a drop in a much longer time after the use of P^{32}, because that is described in the literature. There is one definite advantage of P^{32} over total body irradiation. That is, that the patient receives his dose and does not have to have it repeated for a varying length of time, depending on the blood count. If total body irradiation is used, the patient must have repeated small doses until the desired effect is produced. I would appreciate it very much if Dr. Wright could give us some statistical comparisons, as this is a very important subject.

What I have just said applies only to the chronic forms of these diseases, and I am not considering the acute forms. The acute forms very definitely need something new in the way of therapy.

DR. WRIGHT (closing). I thank Dr. Howes and Dr. Ullmann for their comments. The question was brought up regarding whether we have prolonged the length of life in the chronic leukemias; I would say "yes," if you modify it by "useful life." Whether there is an actual prolongation of life in months or years we do not know, because in both polycythemia vera and in the chronic leukemias the marked individual variation of the disease subjects the statistical studies that had been made to a number of criticisms. We have attempted in our own clinic to do it but I do not think that any valid opinions can be derived from even such an extensive series as that which we have here of 1,100 cases. Opinion must still depend to a considerable degree on clinical impression.

As for polycythemia vera, I don't know. As with the seventy-six year old woman cited, who was treated for thirteen years and then developed an acute myeloblastic leukemia—the question, of course, arises, did we cause her leukemia with the eight years of internal radiation therapy with P^{32}? Yesterday, I saw a young woman only thirty-five years old whom we had been treating for three years for polycythemia vera with P^{32}. Now she is exhibiting in her peripheral blood a scattering of very immature cells qualitatively very suspicious of a chronic myeloid leukemia. Are we producing a leukemia there? The question as to whether the rare terminal leukemia seen in polycythemia vera is a part of the natural course of the disease or whether it is precipitated by a long time internal radiation therapy with radioactive phosphorus remains to be answered. Even if this is one of the rarer potential complications of therapy, radioactive phosphorus is still the most effective therapeutic agent that we have for polycythemia vera at the present.

I would like to make a point about the magnificent work on urethane and nitrogen mustard that Dr. Roy Kracke is doing down at the University of Alabama. They found that urethane and nitrogen mustard are synergistic as antileukemic agents. Though both of these molecules are extremely toxic, their synergism is effective only in their antileukemic action and not in their toxicity. Their work has been done mainly with leukemia in mice. Of course, the effective transposing of this to human clinical material remains to be accomplished but it is an opening and they are making some interesting observations. The knowledge of antimetabolites and cellular inhibitors is rapidly accumulating.

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HODGKIN'S DISEASE*

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IN APPROACHING the problem of the management of Hodgkin's disease, one must first decide what constitutes the aim and goal of proper therapy. If the observer believes that the therapist's function is limited to insuring relief and palliation of symptoms as they disturb the patient, the problem is a simple one and requires no further discussion. If, on the other hand, the observer has concluded that life can be prolonged by active therapy, especially in subacute and chronic forms of the disease, there is much to be said with respect to indications for and methods of treatment.

Since the early days of roentgen therapy, a controversy concerning the proper amount and quality of roentgen radiation has existed. Without going into detail, it would seem justified to conclude that the optimum amount and quality of roentgen radiation is, first, that which most nearly causes a reversion of the clinical and hematologic equilibrium to normal and, second, that which prevents recurrences of disease in areas which are so situated as to make possible "adequate" therapy in patients capable of responding favorably to roentgen irradiation. My associate, Dr. Morton, will discuss further what is considered to be "adequate" therapy in the specific areas and under the conditions encountered.

Not until the development and use of nitrogen mustard therapy was an additional topic added to the controversy between the "palliation" school and those who believe in active, prompt and militant therapeutic management. The philosophy and thinking involved in the decision as to when and how to use alternate courses of roentgen irradiation and nitrogen mustard therapy are motivated by the same reasoning that was invoked during the era before nitrogen mustard therapy with respect to roentgen irradiation alone.

Because of insufficient information concerning the mechanisms involved, it is recommended that substances (folic acid, pyridoxine, etc.), which increase the number of experimental animals surviving an MLD/50 of radiation or chemotherapy, be omitted from the therapeutic regimen before and during treatment. The possibility that one or more of these compounds may aid in protecting the cancer cell against the harmful effects of radiation and chemotherapy is suggested.

RESPONSE TO THERAPY

A number of factors materially affect the response of the patient to therapy and for this reason deserve consideration in detail. In general terms, these factors are (1) the site of localization or principal area of disease involvement, (2) the rate of progress of the disease, (3) the stage of the disease at the time of therapy, (4) the degree of dissemination, (5) the age of the patient, and (6) the presence or absence of unfavorable extrinsic influences.

(1) Presupposing that roentgen therapy is prompt and adequate, superficial and palpable lymphadenopathy in most cases can be managed with comparative ease. The problem of mediastinal lymphadenopathy, when properly and promptly handled (soon after its appearance and before pressure symptoms develop), is not much more difficult, presumably because it can be readily visualized and followed by means of serial roentgenograms. In the absence of secondary infection and other unfavorable conditions, peribronchial lesions and those in the parenchyma of the

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lung may be treated successfully with roentgen irradiation. When one is confronted with therapeutic problems involving the abdominal cavity and the liver and spleen, the outlook changes appreciably. Because of the difficulty in determining the presence of and in localizing lesions before they have become extensive and multiple, the radiologist is forced to use rather large fields with a consequent reduction in total dose; there may result, in addition, widespread destructive changes in nearby and overlying organs and sites which may lead to a disturbance in the over-all physiology of the organism. Because of this difficulty one is forced to rely on directing the therapy at areas most frequently observed at postmortem to be the site of disease; under these conditions the periaortic and retroperitoneal lymph nodes are rather frequently encountered localizations and foci of disease. A reasonably accurate decision concerning the extent of disease activity in the liver and spleen may be made on the basis of the degree of enlargement of these organs, on the basis of laboratory evidence of the impaired hepatic function, or on evidence indicating that splenic hypersequestration of the formed elements of the blood is in progress.

Symptoms as a result of disease within the abdominal cavity should alert the physician to the possibility that he may be confusing involvement in the periaortic and retroperitoneal region with disease affecting central or peripheral nerve fibers in the vertebral bodies and along the gutters and with epidural and subdural involvement. For this reason, abdominal and back pain resulting from Hodgkin's disease should be carefully studied to determine, if possible, its site and mode of origin. To accomplish accurate localization, one must carry out careful roentgen study of vertebral bodies and a complete neurologic examination, including a myelogram and spinal fluid examination if indicated. The demonstration of retroperitoneal adenopathy as a cause of pain, therefore, is made in some cases by eliminating other possible causes which may be confused with it. Despite the use of the most expert methods currently available for the demonstration of adenopathy which displaces other organs and tissues within the abdominal cavity, treatment of disease within this cavity remains unsatisfactory in most cases. Treatment of the spleen and to a lesser extent the liver with roentgen irradiation is carried out by a number of therapists but for the most part does not result in remissions of satisfactory duration as compared with other areas mentioned above. Roentgen therapy over the liver, if undertaken, requires small daily doses and usually does not result in improvement except in very chronic cases. The need for a large triangular field, no larger than the anatomic size of the liver is suggested for this procedure.

In the case of bone and skin, radiotherapy is usually a satisfactory therapeutic medium if the lesion is not widespread and extensive. Large ulcerating tumefactions which arise in the skin and become secondarily infected occasionally do not respond to roentgen irradiation. Because of the many different types and varieties of skin lesions encountered, one is not justified in treating them all in the same manner or in expecting similar results. As a result of the large variety of morphologic and clinical variations in skin lesions observed, it is not uncommon to discover that the physician has attributed the skin lesions present to causes other than the disease itself.

(2) The rate of progress of Hodgkin's disease with respect to its classification as acute, subacute or chronic is of the utmost importance in predicting the extent of response to therapy. In general, the most chronic disease responds best and the most acute least or not at all, with gradations of response between the two extremes. One may judge the presence of unfavorable signs suggesting acuteness rather than chronicity by (a) the shortness of the interval between the demonstrated need for the administration of therapy, (b) the presence of rapidly extending widespread

disease and the presence of systemic symptoms such as fever, night sweats and pruritus during the initial stages of the disease, (c) the existence of unfavorable alterations in hematologic equilibrium early in the course of the disease (leukocytosis, altered monocyte/lymphocyte ratio, anemia and rapid sedimentation rate), (d) the presence of lytic rather than blastic bone lesions, (e) the failure to exhibit weight gain and other signs of physical improvement following therapy, and (f) early clinical evidence of abdominal involvement including hepatomegaly.

(3) The stage of the disease at the time of therapy may be arbitrarily considered as early, intermediate, advanced or terminal. The success of therapy, as a general rule, varies inversely with the lateness of the disease.

(4) Closely allied to the discussion concerning the stage of the disease is the degree of dissemination and the length of time which has been allowed to elapse subsequent to the development of symptoms, signs or laboratory findings indicating an exacerbation. In general, if adequate treatment has been withheld for long periods or if the disease is widely disseminated, or both, a satisfactory response to therapy can be expected only in exceptional cases and those in which the progress of the disease is slow.

(5) A number of unfavorable extrinsic influences have been noted by us in the course of observing patients with Hodgkin's disease. Cellular inflammation and repair such as that resulting from burns, broken bones, contusions, surgery, childbirth and almost all infectious processes (particularly those of a chronic nature, such as abscesses) have resulted in exacerbation and heightening of the rate of the disease process in many cases. Profound physical exertion and fatigue appear to produce a similar effect in some cases. The effect of ultraviolet light and other extrinsic influences which appear to result in increase in the size of the lesions (as a result of edema, proliferation of cells, etc.) is most readily appreciated in lesions involving the nervous system. For example, when the lesion is within the vertebral canal, profound disturbances such as paraplegia may result. Destruction of nerve pathways may also be a serious problem in cases in which extensive edema and local reaction about the Hodgkin's lesion follows roentgen therapy.

We have frequently observed that pain occurs or, if already present, is greatly increased in areas of disease activity a short time following the imbibing of alcohol-containing drinks or foods. This observation may be interpreted to suggest that edema of the lesion may be present under these conditions. It is of interest to note that when a sufficient consumption of alcohol is achieved, the pain is replaced by what appears to be anesthesia similar to that resulting from hypnotic drugs. Whether alcohol produces a deleterious effect on the course of Hodgkin's disease is an intriguing question which remains to be answered. One infectious process which has been observed to produce a temporarily favorable effect in selected cases of Hodgkin's disease is virus hepatitis. This observation is of academic interest only since the phenomenon is not observed in every case and since the risk inherent in the artificial production of virus hepatitis does not justify its routine use.

(6) The age of the patient has a bearing on the response to therapy when one considers patients of all ages as a group; since such wide variations in individuals (of all ages) exist, this consideration may appear of academic rather than practical importance. In general, the outlook appears to be more unfavorable in children.

We have not been able to correlate the response to therapy or the clinical course of the disease with the histopathology of the biopsied lesion.

INDICATIONS FOR THERAPY

In most cases the choice between roentgen irradiation and nitrogen mustard therapy is a relatively easy one. The fact

that the patient has recently had one or the other therapy often, but not always, suggests the choice of the other. When symptoms, signs and laboratory findings indicate the presence of generalized disease or when systemic symptoms constitute the primary consideration, nitrogen mustard is the treatment of choice. When the disease is localized or when local disease appears following a course of HN_2 or following previous roentgen irradiation of other areas, roentgen irradiation is in most cases the treatment of choice. The most common error made by those who treat this type of case is the deferring of HN_2 therapy until the patient is no longer capable of responding to roentgen irradiation. The practice of withholding HN_2 in the presence of widespread disease may permit a number of unfavorable changes to take place. Since the stage is reached rather quickly in many instances when individual adenopathy responds favorably to roentgen therapy although systemic symptoms and abnormalities in hematologic equilibrium do not, the disease may continue to become more disseminated while local areas are being treated. As a result, roentgen irradiation of limited areas at this stage constitutes only partial treatment of the disease and does not produce even temporary total remissions. A number of radiotherapists have maintained that there is a distinct advantage in pinpointing (i.e. the use of very small fields) all observable adenopathy with roentgen irradiation in the presence of disseminated disease. It is true that this technique in advanced cases does not depress the production of the formed elements by the bone marrow as much as the use of more widespread irradiation; however, in advanced disease there are few individuals whose palpable and visible adenopathy represents a large enough portion of their total disease to make possible systemic improvement as a result of the eradication of these accessible lesions.

Let us consider then those symptoms, signs and laboratory findings which are more or less specific indications that roentgen irradiation or HN_2 therapy is needed. A common error in the category of therapeutic indications is the interpretation that pain alone is not a sufficient indication for therapy without the presence of a palpable or demonstrable mass. This mistake in judgment may lead to serious trouble since the longer therapy is delayed, the more difficult it is to obtain a successful result at a later date. In some cases, as a result of destruction or obstructive changes due to the disease, therapy of all kinds is no longer feasible. In general, pain of any kind that is becoming worse daily and that is more severe at night should be thoroughly investigated in an effort to determine its anatomic site of origin.

The presence of any untreated nontender or tender adenopathy or mass(es) or splenomegaly or hepatomegaly is a clear indication for therapy. Anorexia, weight loss, weakness and fatigue are in most cases indications that the disease is moving out of control and requires additional treatment. Systemic symptoms such as fever, night sweats and pruritus call for nitrogen mustard therapy except in certain early cases and under exceptional circumstances in which HN_2 is contraindicated. Cough and dyspnea and symptoms and signs of the presence of chest fluid indicate the need for immediate investigation of lung physiology. It has been found more difficult to treat disease within the thoracic cage, as elsewhere, when a well developed secondary infection is already present. Although a somewhat similar observation has been made in the case of pernicious anemia and diabetes mellitus, the reason for the occurrence of this phenomenon in the three diseases mentioned has not been given.

When a secondary infection in the lung develops as a result of obstruction along the bronchial tree, one is inclined in some cases to use a combination of HN_2 and antibiotics followed in six or eight weeks by roentgen irradiation of the involved area. The choice of HN_2 under difficult circumstances such as those described is based on the fact that it can be used more than once,

whereas our experience suggests that maximum benefit is achieved only once when roentgen irradiation is used in adequate doses in each area and when there is no coexistent secondary infection present.

The presence of disturbances in gastrointestinal physiology (rarely due to intrinsic lesions), the presence of skin lesions (especially the maculopapular variety that occasionally become large, red and ulcerating tumefactions), the presence of pathological fractures and disturbances in sensory and motor function indicate the need for immediate additional treatment. Edema of the extremities is a very troubling and usually a late development in Hodgkin's disease. Its occurrence prior to death in this disease is not invariable and in chronic cases is occasionally capable of being successfully managed over relatively long periods of time.

The demonstration by roentgen studies of lytic or blastic or "mixed type" bone lesions, intrathoracic masses, pulmonary consolidation, atelectasis or pleural fluid, extrinsic pressure on the gastrointestinal and genitourinary tract and abnormalities within the spinal canal demonstrated by myelography are clear indications for immediate further study or therapy as the case may be.

One of the most important and most neglected areas of study in the care of the patient with Hodgkin's disease involves a careful serial analysis of the hematologic status at regular intervals. Though a number of alterations occur, the most reliable indications that the disease is progressing or that unfavorable changes are taking place are the development of lymphopenia, monocytosis, leukocytosis, elevation of the sedimentation rate, anemia, alterations in differential bone marrow estimations and the appearance of myelocytes, group C, in the peripheral blood (usually a late finding).

Serial estimations of the degree of elevation in the basal metabolic rate may offer an additional and helpful means of following the rate and progress of the disease in some cases. Single estimations of the basal metabolic rate are of no more value than single chest films in contributing information to the over-all analysis of the problem and in charting the course of the disease.

To summarize and, if I may, interpret what has been discussed during these past few minutes, the diagnosis and management of the Hodgkin's patient should not stop with the reading of the biopsy section and the first attempt to treat the disease. It should be a persistent and continuing program such as the management of the diabetic patient who requires both diet and insulin. If one relies on a subjective evaluation by the patient with Hodgkin's disease and a cursory physical examination by the physician, complications more serious and irreversible than acidosis and coma can be expected.

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DISCUSSION

Dr. Maurice Lenz, New York, N. Y. Dr. Hoster's excellent paper on the management of Hodgkin's disease emphasizes that correct treatment must be based on familiarity with the clinical aspect of Hodgkin's disease. According to Dr. Hoster, treatment is either symptomatic or active, depending upon the observer's estimate of the function of therapy in the individual case.

We limit vigorous local roentgen therapy to the rare case which, on admission, is clinically confined to a single focus. Treatment should be symptomatic when three or more sites of involvement are clinically recognizable. In these cases, the disease is probably generalized and is incurable by local roentgen therapy. Dr. Hoster emphasized that "pinpointing" vigorous roentgen therapy to clinically involved regions in generalized cases is not worth while; regression of irradiated masses in this stage has less bearing on the progress of the disease than the natural clinical course of silent, untreated foci, which are always present in these cases. Efforts to overcome this shortcoming by irradiating the entire body with roentgen rays or radioactive isotopes fail, since the tolerated dosage is inadequate to destroy completely all foci of Hodgkin's disease. The treatment has to be re-

peated, further reducing tolerance, and finally has to be discontinued. The same difficulty arises with the use of nitrogen mustard—which we employ only in generalized cases and at times alternate with courses of roentgen therapy. Most patients with Hodgkin's disease still come in with extensive involvement because of delayed diagnosis. We reviewed the records of 133 patients with Hodgkin's disease, treated by roentgen therapy at the Presbyterian Hospital, New York, between 1918 and 1940. The interval between initial symptoms and admission was nine months, when patients came in with a single clinical site; it was twenty months when three or more sites were noted. Unfortunately, only one-quarter of the patients came in with one clinical site. Twenty-seven per cent of those with one site and only 4 per cent of those with three or more areas of involvement were free from clinical evidence of Hodgkin's disease, ten years after initial roentgen therapy.

Dr. Hoster has found that invasion of cervical and mediastinal lymph nodes may be handled equally well. This does not correspond to our experience. Freedom from clinical evidence of Hodgkin's disease ten years after roentgen therapy, was noted in 17 per cent of patients with a primary clinical site in the cervical lymph nodes and in none of those clinically originating in the mediastinum. Dr. Hoster correlates fever and loss of weight primarily with extensiveness of the disease. We found these symptoms in three-quarters of the cases which at postmortem examination showed involvement of retroperitoneal nodes. Invasion of this region should therefore be suspected, and its treatment considered, especially if there are also abdominal symptoms or a mass. Clinical involvement of the spleen was noted in about one-half and of the liver in about one-quarter of our cases, and as mentioned by Dr. Hoster, usually signified far advanced disease.

DR. MERRILL C. SOSMAN, Boston, Mass. I would like to ask Dr. Zanes if there is any evidence of synergistic action between the nitrogen mustard and irradiation?

DR. U. V. PORTMANN, Cleveland, Ohio. I would like to ask Dr. Zanes a question that is facetious. He does not have to answer it. I have often suggested to patients with Hodgkin's disease and leukemias who have anorexia that they take some beer or cocktails before mealtime. I just wonder if I have caused exacerbation of symptoms in some of these patients?

Dr. R. P. ZANES (closing). Dr. Sosman, we reviewed our first hundred cases of Hodgkin's disease treated with nitrogen mustard with that question in mind. The results demonstrated that some individuals who had obtained no relief from radiation therapy previous to a course of HN_2, later experienced remissions following roentgen irradiation to a local recurrence of the Hodgkin's disease. So-called "radioresistance" is difficult to evaluate but we concluded that sensitivity to irradiation was enhanced to some degree in these patients by nitrogen mustard therapy.

Dr. Portmann, I think that if you wanted to conduct a research study along those lines, you would not lack volunteers. We have observed that even the small amount of alcohol in a sherry wine gelatin dessert is sufficient to produce severe pain when disease activity is present. This may be true even though no pain exists in the absence of such a stimulus. The ingestion of alcohol may also cause an immediate exacerbation of other symptoms such as itching or cough in these individuals. Only minute quantities of alcohol are necessary and the reaction occurs immediately.

VOL. 64, NO. 6

DIAGNOSIS AND TREATMENT OF LYMPHOBLASTOMA AND LEUKEMIA FROM THE STANDPOINT OF THE RADIOLOGIST*

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TO ATTEMPT any fundamental exposition of lymphomata and leukemia within this time limit would be presumptuous, but certain radiological points of view are thought useful enough to merit emphasis.[6] These diseases have in common their location in the reticuloendothelial system, unexplained fundamental etiology, and variable course to a fatal termination. With our present knowledge, therapy is limited to the application of various potentially toxic agents in the hope of securing a differential inhibition of the disease without intolerable damage to normal tissues. Accurately administered radiation therapy continues to be the mainstay in the control and is especially useful when the disease is localized. Another aspect of therapy is the management of a series of complications which result both from impaired resistance to infection and from interference with metabolism. Such interferences are brought about by the reticuloendothelial location of the disease plus lack of "specificity" of therapeutic agents for the involved tissue with eventual undesirable side reactions.[3,8]

Patients with leukemia and Hodgkin's disease should be treated by their local medical groups whenever possible. The home environment is more economical and humane, simplifies the patient's adjustment to his disease, and frequently makes possible at least a part-time gainful occupation. Futhermore, with access of patient to physician, early reactivation or complication can be discovered before it extends beyond control. Unless the diagnosis is unusually obscure, optimum management can be achieved by cooperation between a local or regional group consisting of internist-hematologist, pathologist and radiologist. Radiological service is available in most communities combining both the diagnostic and therapeutic aspects of the dual speciality, and in this group of diseases such a combination can be most efficient. Diagnosis influences therapy to such an extent that only the closest correlation of diagnostic and therapeutic radiology can keep unpleasant symptoms to a minimum.

The diagnostic radiologist has the initial problem of differential diagnosis of the presenting hematological symptoms, whether the initial disturbance lies in the marrow or other portions of the reticuloendothelial system. Choice of bone for aspiration biopsy may be guided by the presence of destruction or other variation. This is particularly useful should the lesions consist of agrregations rather than diffuse infiltration as in some types of multiple myeloma,[1] metastatic tumor, or leukemia.[10] Roentgenographic survey will usually indicate the most efficient diagnostic approach; either aspiration biopsy, exploratory operation, or other means of laboratory or clinical differential diagnosis. Diagnosis exclusively from a single laboratory or roentgenographic procedure belongs to an earlier era. My colleagues have disagreed with this statement, claiming that diagnosis by biopsy in some lymphomas is quite reliable. However, we question this infallibility and have seen initial or working diagnoses in error in several situations. Leukemoid reactions following

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bone marrow irritation from infection and tumor have been confused with myelogenous leukemia. Virus infections producing lymph node enlargement and/or peripheral lymphocytosis have similarly been confused with lymphoblastoma and lymphatic leukemia. We have also observed benign granuloma originating from chronic infection, metabolic dyscrasia, or abnorml allergic reactions such as sarcoidosis being confused with lymphoblastoma. Variations within the lymphoblastoma group from node to node and through the course of the disease must be accurately interpreted as they alter therapeutic indications. In the discovery of the cause of a toxic marrow depression, roentgenographic and other methods can be employed successfully in locating tumors and infections. Errors of diagnosis and management can best be avoided by combined efforts of a group of physicians considering all the aspects of the disease.

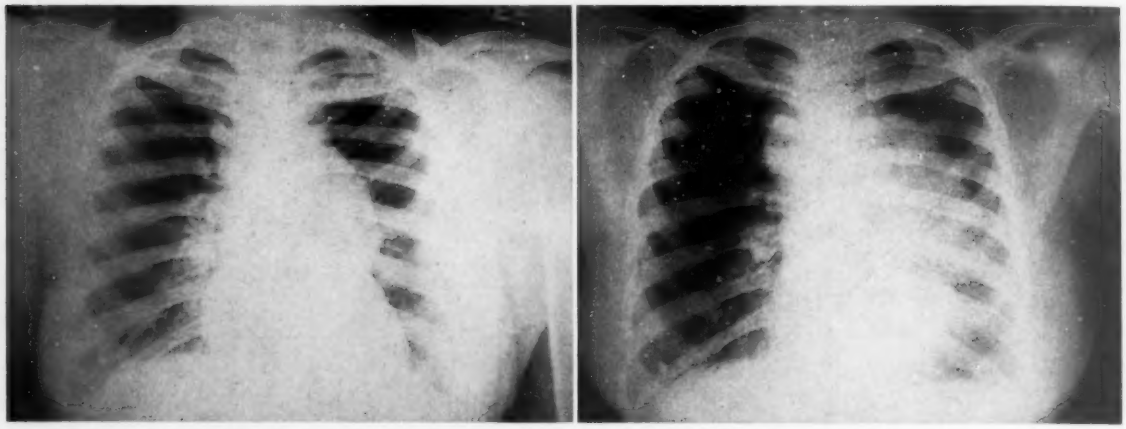

FIG. 1. L. B. A young colored girl with Hodgkin's disease requiring thoracotomy for definitive diagnosis. Note increase in size of lesion during eight month period when tentative diagnosis was tuberculous lymphadenitis. Patient originally asymptomatic with discovery of mediastinal mass on routine examination for food handler's permit.

Diagnosis by irradiation, the so-called "therapeutic trial," is mentioned to be condemned. Such a practice hinders definitive therapy later and can produce injury in certain infectious diseases. When the condition requires thoracotomy or laparotomy for tissue biopsy, the lesion is sufficiently well localized that cure or long-term palliation may result from aggressive therapy (Fig. 1). The opportunity for long-term control is lost while waiting to determine if the condition regresses sufficiently to be considered a lymphoblastoma. The phenomenon of radioresistance in early lymphoblastomatous disease appears to be the direct effect of inadequate initial therapy. With present advances in anesthesia and improvement in surgical techniques and judgments, the risk of biopsy is probably less than not knowing when or where to treat the disease. Our best results in nonleukemic lymphomata are obtained by the application of at least 2,000 roentgens to the tumor mass and contiguous lymphatics, using fields that in so far as possible avoid normal tissue and employing comparatively heavy filtration. A dose of this magnitude may be disastrous in the presence of some infectious diseases and certainly impairs resistance in those situations where radiation is of no proved value.[4] Smaller doses apparently encourage the development of fibrosis and scarring without eradicating the active disease. Such a lesion in the thorax and abdomen leads to exudate from lymphatic block and impairment of vital visceral function.

Slaughter and Craver[11] have commented

upon the frequency with which primary lymphoblastoma originates in the deep nodes. This concept fits the disease as we commonly observe it. Unfortunately, almost without exception, visceral cases are discovered after the disease has spread from its primary focus to the adjacent lymph nodes. Until there is something better than ionizing radiation for the control of local manifestations of these diseases every effort should be made to deliver as effectively directed a dose as possible, beginning with the lesion most likely to incapacitate the patient.

The location of visceral areas involved is difficult, but valuable information can be elicited for the location of treatment portals by roentgenographic examination when carefully done and compared with clinical and laboratory data. The plain film demonstrates splenic and hepatic enlargement. Further confirmation of spleen size and position is obtained by barium enema and upper gastrointestinal series. The spleen is enlarged if on the anteroposterior roentgenogram the thickness excedes 6 cm. and is under suspicion if the product of the horizontal and vertical dimensions exceeds 75 cm. That splenic enlargement can be demonstrated to have occurred without equaling these measurements is illustrated by finding the spleen to be smaller in size on the roentgenogram before or after an acute infection, but never roentgenographically enlarged beyond the stated criteria at the height of infection. Consequently we consider the above measurements to have considerable reliability in the diagnosis of splenic enlargement.

Physical examination utilizing palpation and percussion are subject to inaccuracies due to abdominal wall thickness, variation in position of the diaphragm, and the degree of cooperation of the patient. In rare instances the spleen lies downward and away from the diaphragm, but the relationships to the splenic flexure of the colon from the attachment of the splenocolic ligament are maintained so that the colon lies medially on its pedicle so that it fills the space behind and above the stomach without casting a typical shadow on the anteroposterior roentgenogram. Under these conditions, studies with the patient standing and with the stomach and colon filled with barium will usually demonstrate the size of the spleen as it rotates into the more usual position.

Pneumoperitoneum clearly demonstrates the splenic outline but is not without discomfort and danger. In a single instance, pneumoperitoneum was valuable in differentiating the liver enlargement from pleural involvement where the diaphragmatic position could not be resolved by standard techniques. Splenic and hepatic visualization with thorotrast is found unnecessary except in those rare instances of congenital hemolytic icterus where symptoms persist or recur after splenectomy. The surgeon cannot be expected to find a small accessory spleen without guidance when the critical condition of the patient does not warrant an extensive exploratory operation.

The differential diagnosis of the causes of splenic enlargement roentgenologically is a convenient approach for the radiologist presented with a suspected hematologic problem. In certain instances this is the most expedient method of diagnosis. For example, the finding of esophageal varices, a large spleen and a small liver establishes the diagnosis of Banti's syndrome (Fig. 2). A study of the long bones may show changes typical of Gaucher's disease, such as widening and thinning of the distal femoral cortex (Fig. 3) or fragmentation of the femoral head.[14] Other changes may be less typical but suggest a diagnosis. In chronic myelogenous leukemia there is frequently a large spleen and a haziness of the lung fields due to anemia, thrombi or leukemic infiltrates. Although there is wide variation, the splenic enlargement of chronic lymphatic leukemia is usually less than found in myelogenous leukemia and is associated with lymph node enlargement. In polycythemia rubra vera an enlarged spleen is frequently associated with vas-

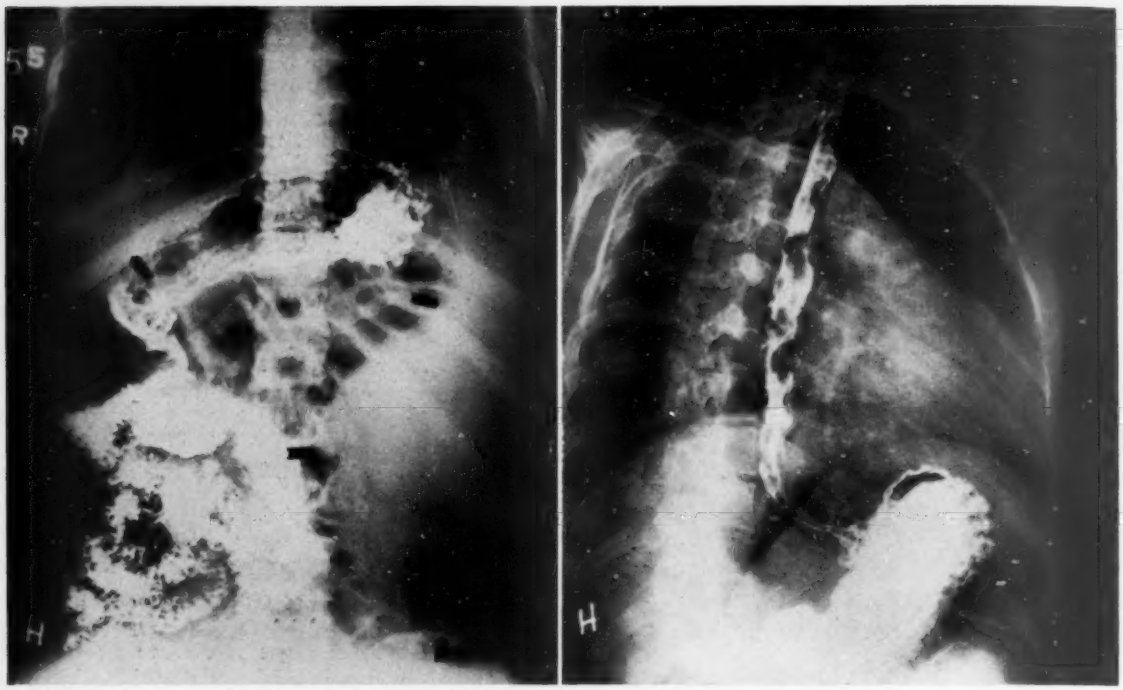

Fig. 2. R. M. Banti's syndrome showing diagnostic triad of large spleen, small liver, and esophageal varices.

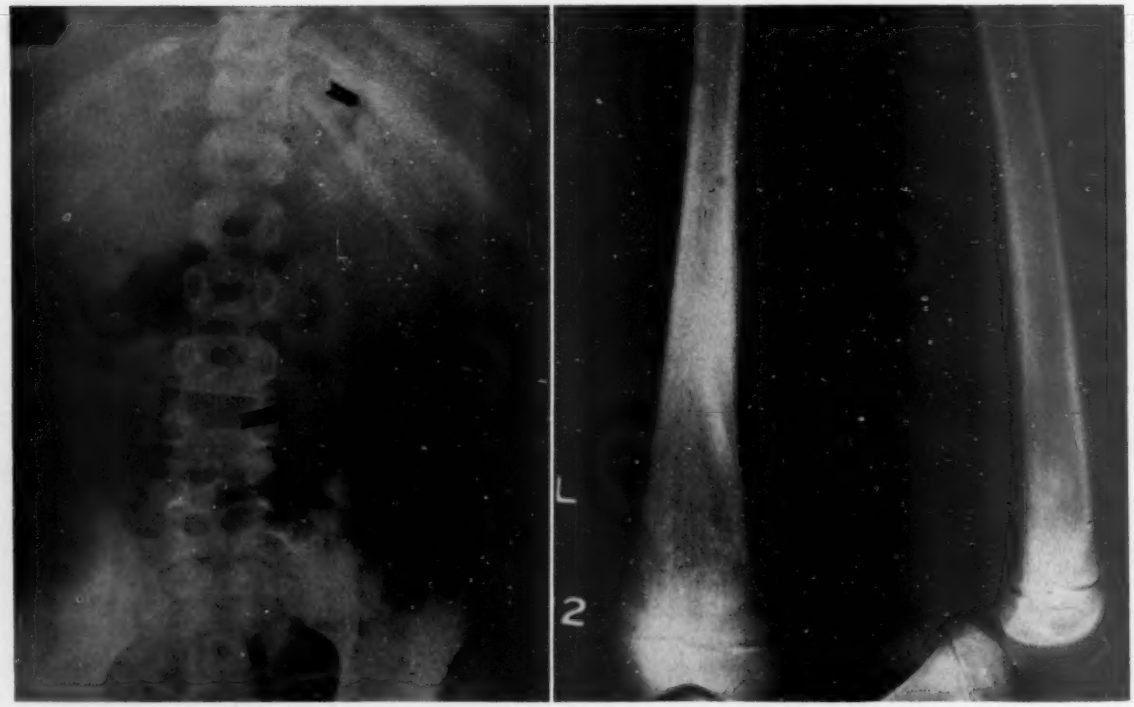

Fig. 3. L. S. Gaucher's disease showing splenomegaly and typical changes in distal diaphysis of femur.

cular engorgement of an almost typical pattern in the lung and occasionally with duodenal ulcer.

Roentgenoscopically the lymph nodes of chronic lymphatic leukemia and the frequently found smooth mediastinal sheath of acute lympholeukosarcoma have a much more flexible appearance with respiration than does the mediastinal infiltration of Hodgkin's disease or Boeck's sarcoidosis. This we have thought to be indicative of a different type of supporting stroma. The smooth obliteration of the mediastinal vascular outline by lympholeukosarcoma, which ensheaths the pericardium (Fig. 4). is almost as typical as some observers consider the "potato nodes" of mediastinal sarcoidosis (Fig. 5). The symmetrical hilar involvement of another type of sarcoidosis may be associated with a characteristic pulmonary infiltration (Fig. 6). Myelofibrosis or sclerosis with myeloid metaplasia in the spleen causing enlargement of that organ may occur as a primary disease or secondary to tumor invasion of the marrow. Long standing cases of myelofibrosis sometimes exhibit increased trabecular calcifications[15] (Fig. 7).

These valuable indications as to probable diagnosis, however, must always be confirmed by microscopic biopsy. The pathologic diagnosis may disagree with the roentgenologic findings. For this reason, the radiotherapist should see the microscopic slide in order to avoid error. There is some merit in varying the rate, dosage, and treatment area from consideration of the microscopic classification of the lesion.[9]

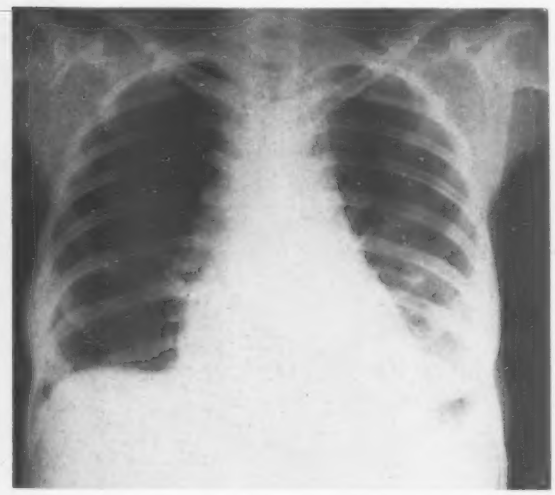

Fig. 4. B. W. Lympholeukosarcoma (synonym, acute lymphatic leukemia) showing loss of mediastinal outline due to infiltration.

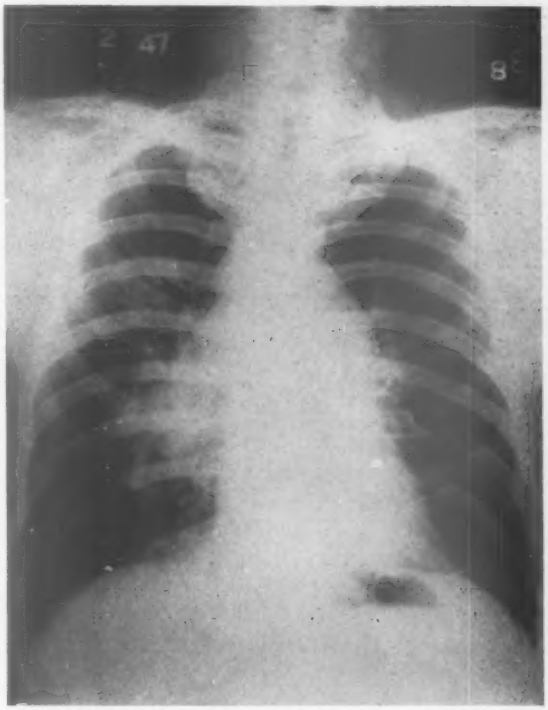

Fig. 5. R. E. No easily accessible nodes available for biopsy. Patient given therapeutic trial of irradiation in another clinic without demonstrable change in size of disease process. Retrogressive course with autopsy verification of Boeck's sarcoidosis.

At times all methods fail to locate the site of renewed activity in Hodgkin's disease and reliance must be placed on deductive reasoning to establish therapy portals especially in abdominal fields.[16] Attention to earlier sites of involvement may indicate extension through the diaphragm from the mediastinum (Fig. 8); or the previous presence of iliac nodes suggests contiguous lower abdominal lymph node enlargement. Early periaortic involvement in the abdomen is probably not discoverable by present methods. Anteroposterior and lateral views may show displacement of calcified abdominal aortic plaques. Excretory urograms

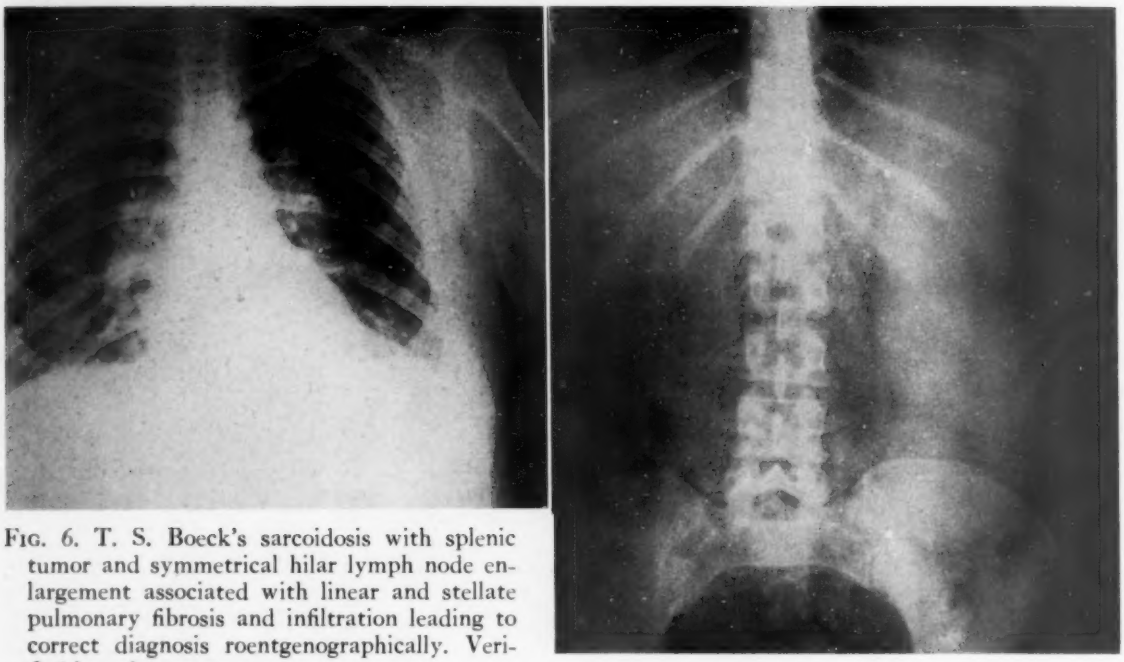

Fig. 6. T. S. Boeck's sarcoidosis with splenic tumor and symmetrical hilar lymph node enlargement associated with linear and stellate pulmonary fibrosis and infiltration leading to correct diagnosis roentgenographically. Verified by splenectomy.

Fig. 7. T. W. Myelosclerosis with large associated splenic tumor.

will show retroperitoneal changes if the oblique and lateral roentgenograms are added to the routine views. Barium studies are worth while, and even cholecystography may be helpful as illustrated in the case of P. L. (Fig. 9).

In early involvement of the mediastinum by lymphoblastoma, pressure studies with the patient blowing into a mercury manometer are helpful. If the pressure is maintained above the expected pulmonary artery pressure, and the hilar or mediastinal shadow becomes more prominent, graphic evidence of lymph node invasion can be secured (Fig. 10 and 11). Regression under therapy of the smoothly outlined lymph node masses should be complete even when the nodes have enlarged to massive proportions. In the instance where the process has extended through the capsule producing

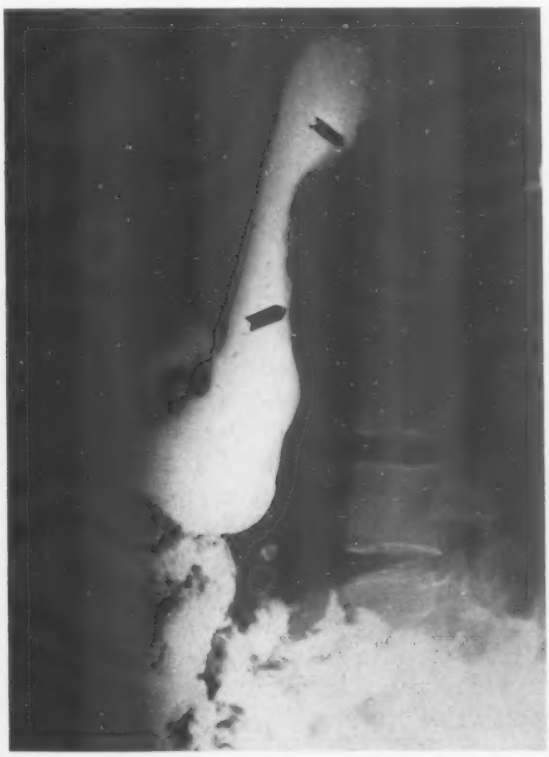

Fig. 8. R. B. Hodgkin's disease discovered in left retrogastric lymph nodes. Recent exacerbation of symptoms without evidence of other active disease in case with initial mediastinal involvement. Therapy directed to this area produced remission of symptoms and disappearance of nodes.

Fig. 9. P. L. Unusual location of enlarged lymph nodes in Hodgkin's disease between gallbladder and duodenal bulb.

peribronchial accentuation or early atelectasis, regression is frequently less satisfactory (Fig. 12). Such peribronchial infiltration indicates neglected late disease or acute disease with a less favorable prognosis. It should be emphasized that despite the tendency of these patients to suffer from secondary infection, pulmonary infiltration persisting after a trial at chemotherapy must be considered a manifestation of Hodgkin's activity and vigorously treated. Procrastination results in irreparable damage to the lung with secondary bronchiectasis.

Classically, early Hodgkin's disease involves lymph nodes, but may rarely occur primarily in bone marrow, liver, spleen, kidney, and supporting tissues. Most bone or nervous system involvement occurs in patients with evidence of previous lymph node disease. This implies that distressing or painful complications may be prevented by early definitive treatment combined with careful follow-up. Once nervous system involvement has occurred, accurate localization is imperative. Myelography of the spinal canal is safer than relying upon clinical localization for a site of infiltration or pressure. Missing the exact location by the width of a treatment portal is easily done when employing clinical methods, and usually insures a paraplegia which will

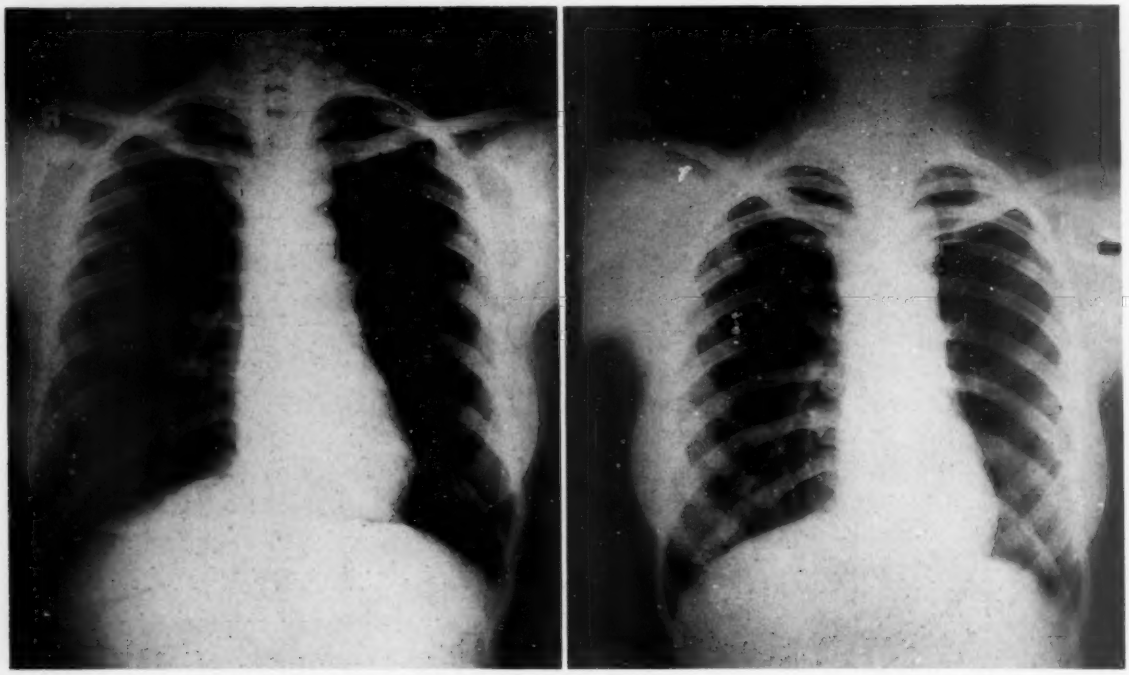

FIG. 10. F. W. Hodgkin's disease of mediastinum suspected on plain film. Infiltration proved by manometer test. Note elevation of mercury column above 50 mm. of mercury which is well over normal pulmonary arterial pressure.

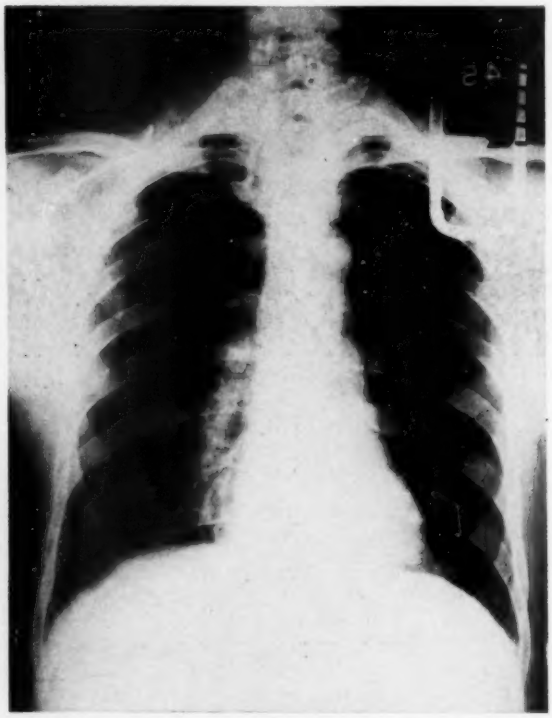

FIG. 11. F. K. Chronic lymphatic leukemic nodes made prominent by increase of intra-alveolar pressure above 45 mm. of mercury.

later fail to respond to treatment. Clinical manifestations of involvement of the spinal nerves after they emerge from the medullary canal can successfully deceive the experienced neurologist and neurosurgeon as to the exact location of the disease and lead to ineffectual treatment.[12]

Pain frequently precedes new tumor formation in the known Hodgkin's case. If the factors of previous location, probable site and method of extension are combined with a careful neurologic examination, portals for radiation therapy are usually found which will control the extension before actual tumefaction occurs.

Little has been said about treatment except in Hodgkin's disease. Localized lymphosarcomatous lesions[2] are treated more intensively up to skin tolerance for the chosen portal area. Management of local complications in leukemia is directed toward specific symptomatic relief without an attempt to control the generalized cellular manifestations. The dosage is widely variable depending upon desired results.

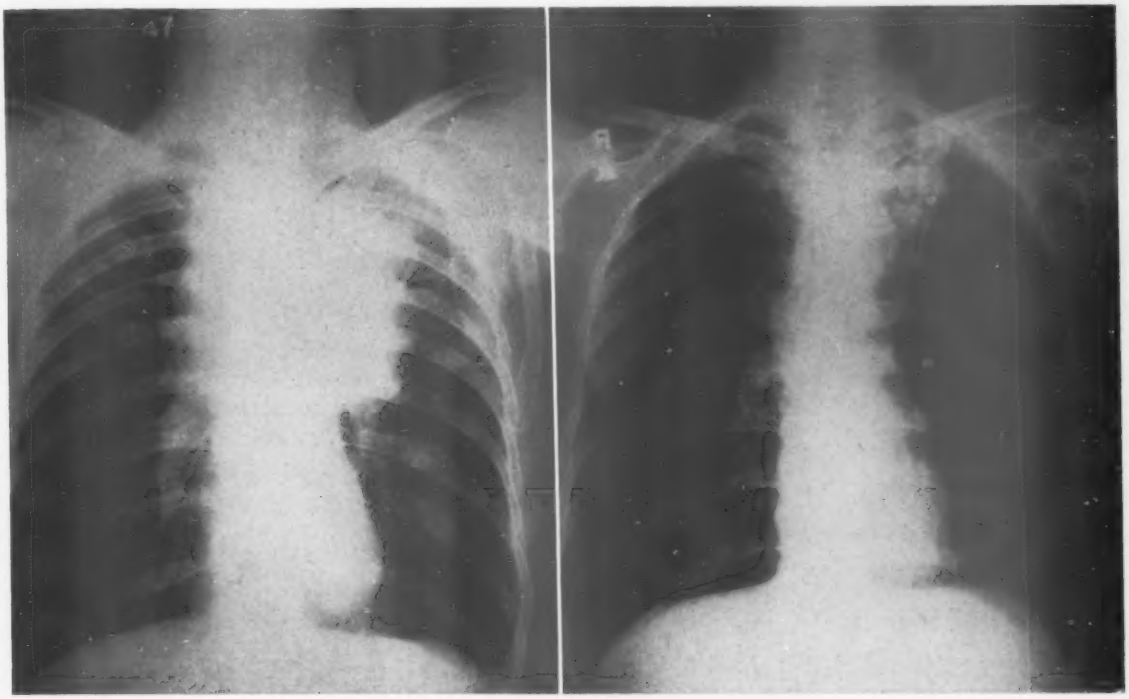

Fig. 12. V. M. Hodgkin's disease with late mediastinal involvement illustrating residual left upper lobe fibrosis unavoidable when lesion has extended beyond capsule of node into lung before therapy.

Daily doses of from 50 to 100 roentgens are advisable until the sensitivity of the white cell response to irradiation has been determined. Caution should be exercised not to over treat, and daily blood counts are essential during therapy. Care should be taken to permit a margin of reserve white cells as the count will usually decline for a few days after cessation of therapy.[6] Giant follicular lymph node hyperplasia is controlled by therapy similar to that employed in Hodgkin's disease.[13]

In summary, we believe that after satisfactory diagnosis is established the patient can best be treated near his home, because complications can be recognized sooner; the patient may work if he is able; he is surrounded by his friends; and continuation of a normal life is limited only by his physical condition. An attempt has been made to outline the importance of the diagnostic radiologist and to suggest that successful management of this group of diseases requires constant integration of both roentgen diagnostic and therapeutic disciplines.

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DISCUSSION

DR. HAROLD W. JACOX, New York, N. Y. In general I am in agreement with Dr. Morton's remarks, and there is not much I wish to add. I, too, have seen lymphoblastoma that was difficult of diagnosis and needed all the aids at our disposal to arrive at a true evaluation. The mimicry of these conditions at times is as great as it is in lues. Dr. Morton is right in emphasizing the importance of making an accurate diagnosis even though occasionally one has to resort to exploration to do it. Only then can the patient be treated intelligently.

DR. WILLIAM HARRIS, New York, N. Y. There is a question that Dr. Morton brought up. Dr. Jacox agreed with him. That is the question of clinical trial with irradiation to make a diagnosis.

Now there are patients with mediastinal masses, particularly in the superior mediastinum where it is impossible to make a clinical diagnosis without thoracotomy. Although thoracic surgery has made tremendous strides and the mortality from exploratory thoracotomy is small, there is still a mortality and I believe that it is quite safe if the patient has been very, very thoroughly studied in every way and you have ruled out inflammatory disease and where your working diagnosis is lymphoblastoma or lymphogranuloma, to use radiotherapy for a clinical trial to help establish a diagnosis.

Just in the last year, 6 patients were explored at our hospital and found to have Hodgkin's disease. In 3 of them, the surgeon tried to remove the disease and felt he had removed it completely. The disease promptly returned. We made one error in the last hundred cases of mediastinal tumor that were treated on this basis, that was a patient with a thymoma which decreased about 33 per cent after receiving 2,000 r. Therefore, I believe in these cases of mediastinal tumor where a clinical diagnosis is not made a therapeutic trial with roentgen irradiation is indicated.

DR. MORTON (closing). I wish to thank Dr. Jacox and Dr. Harris. Personally, if I had lymphoblastoma, I think that I would find a conservative surgeon who would not try to remove it from my mediastinum and have a biopsy. Then I would want treatment until the same regressed completely without any strands projecting into the lungs indicating later bronchitis and fibrosis. We see a goodly number of referred patients who have had 900 roentgens, 1,200 roentgens or less or a roentgen treatment once a week for six months with intractable fibrosis. It is possible to get by, perhaps, with doses of 2,000 roentgens. Yet, on the other hand, I wouldn't like to have 2,000 roentgens delivered with 200 roentgens daily to my mediastinum if I had tuberculous lymphadenitis or sarcoidosis.

Vol. 64, No. 6

THE DIAGNOSIS OF EARLY INTESTINAL CANCER*

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UNDER any circumstances it would be a great pleasure, and a signal honor, for me to participate in a program of the St. Louis Medical Society. Members of this society have made and are continuing to make great contributions to the science and art of medicine. Many of the truly great spirits of American medicine have lived and labored here. The medical world of today continues to look to St. Louis for advance information, for inspiration and for guidance. But to be invited to your midst to assist you in an annual commemoration of the name and spirit of Russell D. Carman, is an honor and a privilege which I deeply appreciate. Unfortunately for me, I did not know the man in life. He had passed away eighteen months before I set foot in the department where his genius matured, after having been nurtured to adolescence and manhood in this community by you and among you. Carman's spirit, however, was very much alive when I arrived on the scene; hallowed and revered by his lifelong friend and associate, Albert Miller—another St. Louisan, by the way, who is still with us, active and hearty on his eighty-first birthday last Friday—and by his pupils, A. B. Moore, B. R. Kirklin, Charles Sutherland, and somewhat later, John D. Camp. All of these men by virtue of Carman's inspiration and guidance have achieved a modicum at least of eminence in their own right. After so many years of intimate association with these men, of course I knew Carman. Steeped in it as I have been for these many years I could not do otherwise than to absorb some of his flavor. I became one of the many who had every reason in the world "to grieve because he died, and to rejoice because he lived."

Carman knew cancer of the gastrointestinal tract. It is sad indeed to consider how intimately Fate had ordained it for him to become acquainted with the disease. I often think, as I refer again and again to his papers, and to the monograph[2] which he and Albert Miller prepared so well, how pitifully little has been added through the years to the knowledge they had acquired about its roentgenologic manifestations. True, a great part of Carman's energy was devoted to the elaboration of a set of reliable criteria by which the disease could be recognized as such in the stomach where its morphologic features are so multiform. How well he succeeded is a matter of history. He did not have time, however, to become as convinced about the roentgenologic manifestations of intestinal cancer as he was about those of gastric and esophageal cancer, similar in almost all respects as we now recognize them to be. A few of Carman's contemporaries were convinced. One has only to read the early papers of such men as Haenisch, and Gottwald Schwarz and Case to appreciate the keen perception of those pioneers who in less than fifteen years after Röntgen's discovery had elaborated and put into clinical practice the important principles and techniques which we use today in the roentgenologic diagnosis of mature intestinal cancer. In those days, however, knowledge of roentgen rays was not disseminated, nor was experience accumulated as rapidly as today, and it required some time for intestinal roentgenologic diagnosis to arrive at its deserved position, parallel to gastric roentgenologic diagnosis, in reliability and in accuracy. For almost a quarter of a century we have had, in the roentgenologic approach, a diagnostic maneuver which can be made to detect and recognize, as such, neoplastic disease of the intestine—

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above the reach of the proctosigmoidoscope—at least as early in its development as it will manifest itself by clinical signs and symptoms. This may seem to be a large statement to make, but it is a thesis which I feel can be defended with ease just on the basis of the record. I made the qualification, "above the reach of the proctosigmoidoscope" because I contend that, for several good and sufficient reasons, the roentgenologic maneuver has not been satisfactory for the diagnosis of rectal and sigmoidal lesions within the reach of this instrument; nor do I believe that it can be made to be satisfactory, and it is a mistake for the roentgenologist to assume the responsibility of diagnosis for that portion of the intestinal canal—wrong for his clinical and surgical consultants to ask him to do so. The proctosigmoidoscopic examination has the obvious advantages of direct vision and contact. It thus reveals much more than the mere form and size of a pathologic process, and makes available instantly material for histologic and bacteriologic study. It thereby gains insuperable advantages over the most competently performed roentgenologic examination in the portions of the intestine to which it is specifically applicable, regardless of refinement in roentgenologic technique and of virtuosity of roentgenologic examiners—always provided, of course, that the proctosigmoidoscopic examination is done with competence and care.

Assuming that the best treatment of intestinal cancer known today is extirpation by surgical methods and that proctosigmoidoscopy and roentgenologic examination are means of detecting cancer of the intestine at a time when it produces the clinical symptoms and signs that bring the patient to the physician, if not well in advance of that time, we should expect that the survival rates after surgical treatment of intestinal cancer should have shown marked improvement in the last ten or fifteen years. I think it can be shown that these survival rates have improved, but statistical studies recently made by Dixon[3] reveal that the over-all five-year survival rate after surgical treatment of intestinal cancer is still just below 50 per cent (Table 1). This figure may be gratifying and encouraging, but it is not one with which to be content. These studies also show that far too many patients, at least 4 of 10, come to operation at a time when demonstrable metastatic spread to regional lymph nodes, liver, or both has already occurred (Table II). In the group of patients without manifest metastatic lesions at the time of operation, the five-year survival rate was 62 per cent. Unquestionably, some of the patients in the group last mentioned failed to accomplish five-year survival because their metastatic lesions were so minute at the time of exploration that they escaped detection. Obviously patients must somehow get to

TABLE I

FIVE-YEAR SURVIVAL RATE IN 2,103 CASES OF MALIGNANT LESIONS OF THE LARGE INTESTINE IN WHICH RESECTION WAS PERFORMED; 1931 TO 1940 INCLUSIVE

Grade of Malignancy	Total		No Metastasis		Metastasis	
	Patients	Survival, per cent	Patients	Survival, per cent	Patients	Survival, per cent
1	350	66	281	73	69	35
2	1,307	50	803	61	504	32
3	317	36	126	52	191	26
4	106	26	31	35	75	23
Not stated	23	43	15	40	8	50
Total	2,103	49	1,256	62	847	30

the physician earlier in the course of the development of their neoplastic lesions, but this will accomplish little unless the physician learns to extract the maximal yield from the diagnostic maneuvers now at his disposal to discover the lesion when it is small and treat it at that time.

Perhaps some day a test or a clinical syndrome will be elaborated which will reveal or specifically indicate the existence of internal cancer before the disease has attained macroscopic proportions. That day has not yet come. Whether gross pathologic, proctosigmoidoscopic, or roentgenologic, our diagnosis is, by and large, limited—with the exception of the recognition of cancer cells in excretions, secretions and exudates—limited to the recognition of certain characteristic gross morphologic changes in the affected organ. How, then, does early intestinal cancer look? Let one experienced in gross pathology and in surgical pathology say what he thinks a representative opinion of experts in those fields would be. I quote from a personal communication from Baggenstoss:[1] "Cancer of the intestine usually is manifested in its earliest recognizable stage of development as a polyp. Such a lesion may be sessile or pedunculated and the earliest histologic signs of malignancy may be present in its entire epithelial surface or only in a portion thereof. Theoretically intestinal cancer may also begin as a tiny focus of malignant cells in otherwise normal epithelium. This is known as 'carcinoma in situ.' This focus may not be visible macroscopically and may be too small to be palpable, and then it is discovered only by fortuitous histologic section. Theoretically, also, and reasoning by analogy from the occurrence of such lesions in the stomach, intestinal cancer may also arise in a previously benign ulcerative process. Such solitary benign ulcers occur very rarely in the colon, and I have never observed an instance in which malignant neoplastic change occurred in the edges of a benign intestinal ulcer." Not to be inferred from this statement is the idea that all polypoid lesions of the intestine are or will become cancer. It does imply that some of them are cancer, that many of them will be cancer if their bearer lives long enough, that most cancers of the intestine begin as lesions which have the gross pathologic features of the lesion called a "polypoid lesion," and that there is no sure way, short of microscopic examination—not of a part but of all of a lesion of that kind—of distinguishing the benign from the malignant or potentially malignant lesions.

Table II

INCIDENCE OF DEMONSTRABLE METASTASIS IN 2,103 CASES OF MALIGNANT DISEASE OF THE LARGE INTESTINE IN WHICH RESECTION WAS PERFORMED 1931 TO 1940 INCLUSIVE

Grade of Malignancy	Cases	Metastasis Demonstrable		Metastasis Not Demonstrable	
		Number	Per Cent	Number	Per Cent
1	350	69	19.7	281	80.3
2	1,307	504	38.6	803	61.4
3	317	191	60.3	126	39.7
4	106	75	70.8	31	29.2
Not stated	23	8	34.8	15	65.2
Total	2,103	847	40.3	1,256	59.7

In the overwhelming majority of instances polypoid lesions 1 cm. or so in diameter, and many larger ones, are easily removed by transcolonic excision. A great many of the lesions of this size occurring in the rectum and lower portion of the sigmoid colon are successfully extirpated by fulguration and with radium therapy. The neoplastic intestinal lesion 1 cm. in diameter or slightly larger may be the source of metastasis to the regional lymph nodes or to the liver, or to both, but this is not a common occurrence. Certainly for therapeutic purposes the smaller such lesions are the better, although not all large ones are ineradicable because of metastasis and metastasis may originate in the smallest lesions (Fig. 1 and 2). In a general way, it is also true that the smaller and more circumscribed a lesion is, the less is its ability to cause clinical symptoms and signs. It is a rare experience to encounter

an intestinal lesion 1 cm. in diameter which shows a high grade of malignancy.

While it is true that the great majority of polypoid and other intestinal carcinomas are of a low grade of malignancy a significant number of patients die from the effects of these lesions. The grading of neoplastic lesions is, for practical purposes, chiefly an expression of the rate of their growth. There would be no purpose of attaching the appellation "malignant" to lesions of grade 1 if experience did not consistently show that they take a definite but relatively small toll of life. A patient who dies of metastatic cancer five or more years after extirpation of the original growth is just as dead as the one who dies within a year.

The fibroscirrhous annular cancer of the intestine, the gelatinous cancer partially or completely encircling the intestine, the medullary ulcerating mass protruding into the intestinal lumen,—none of these was always of that size and of that configuration. Presumably they began as single cells or as relatively small groups of cells which may well have existed at the site of the subsequent development of definite cancer at birth or even before that time. When favorable conditions arose they began to proliferate. Gradually their mass expanded from microscopic to macroscopic proportions and up to the point where they could be seen with the naked eye. This is the stage in the pathogenesis of an intestinal neoplasm at which it can be detected with modern clinical diagnostic methods. The lesion would be described as a "polypoid lesion," a small excrescence on the surface of the mucous membrane of the intestine. We have no way of knowing how such small lesions make the transition from the stage of "polypoid lesion" to the stage when it assumes the more familiar gross

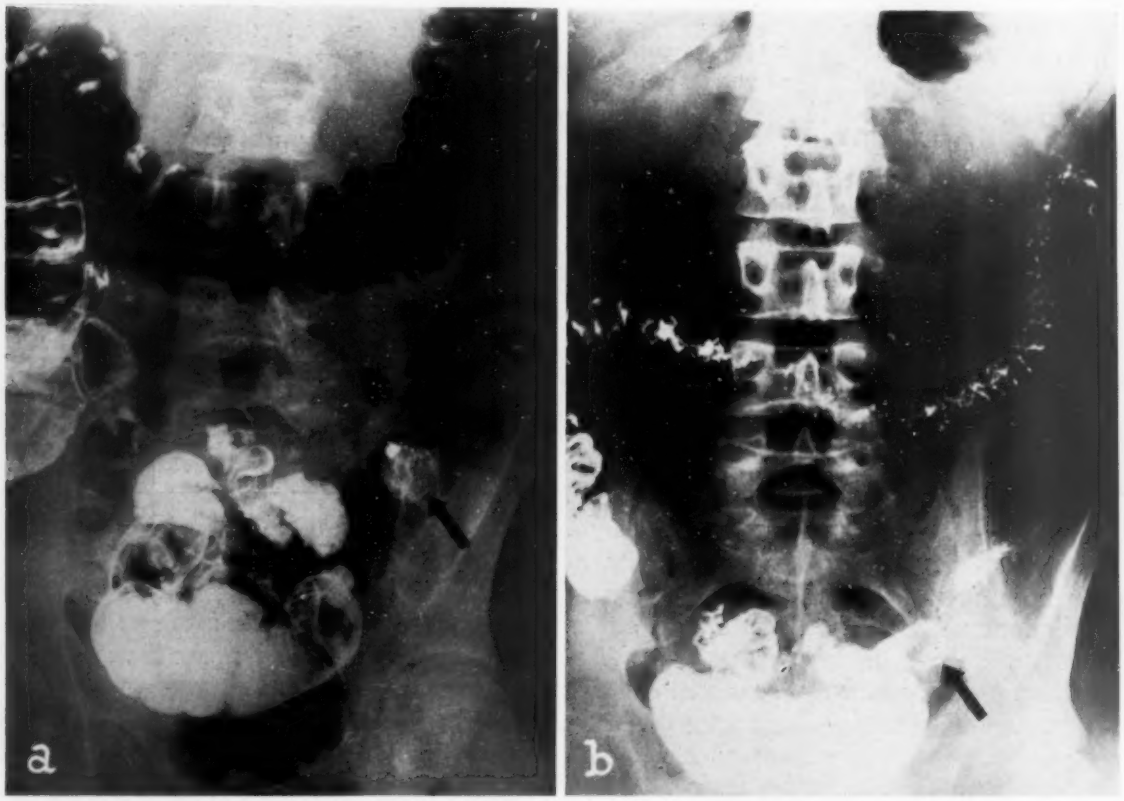

FIG. 1. Pedunculated polypoid lesion, 4 cm. in diameter, low in the descending portion of the colon. *a*. A double contrast preparation was used. *b*. Portion of colon after expulsion of contrast enema.

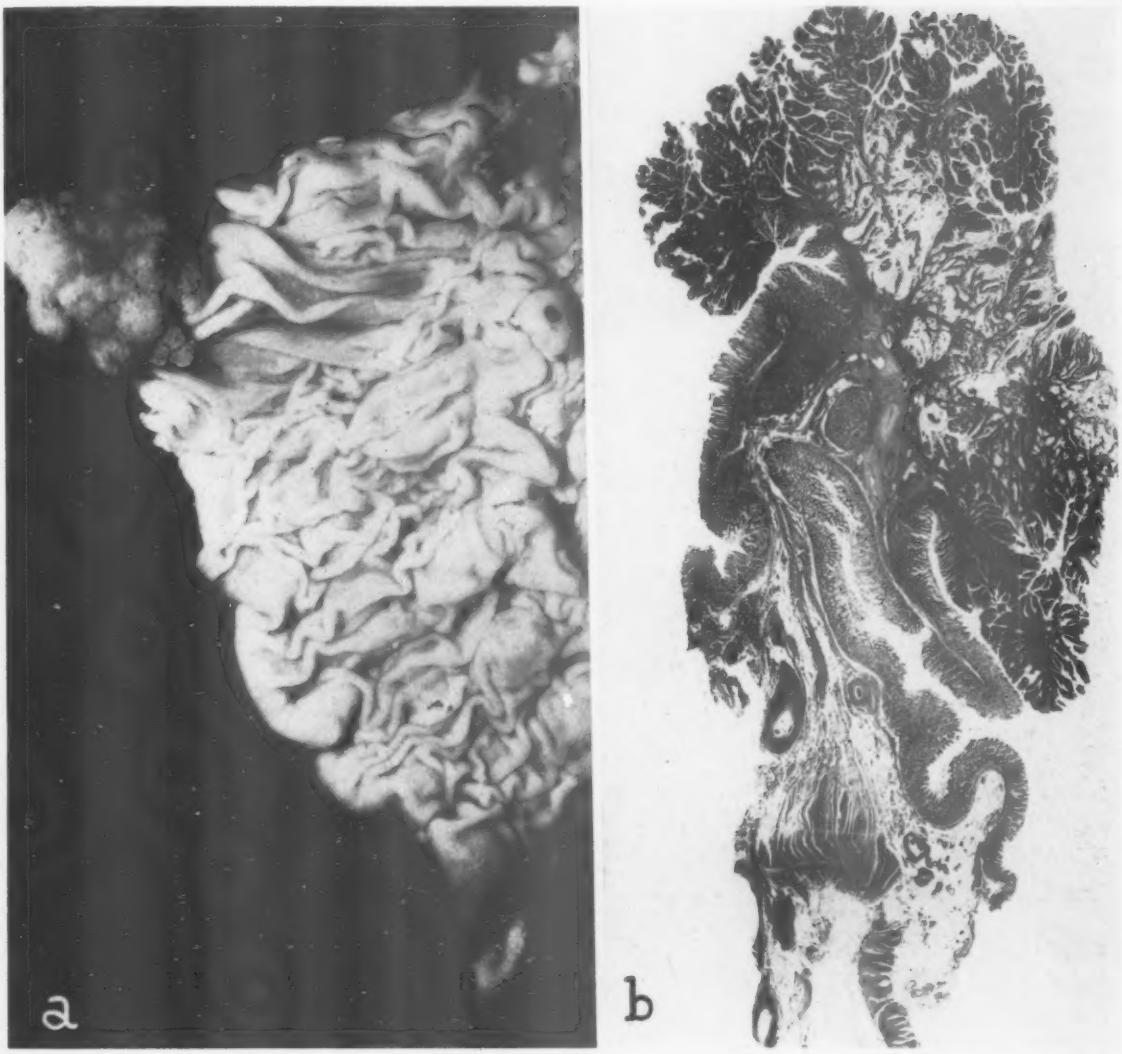

FIG. 2. *a*. Resected specimen. *b*. Magnified section of the neoplasm. Microscopic examination revealed adenocolloid carcinoma, grade 2, "in situ" in a tiny portion of the lesion. At least one regional lymph node was found to be similarly involved. Operation was performed in 1933; the patient was alive and well in 1949.

pathologic features of well-developed frank cancer. I have had the opportunity to observe such a transition in several instances (Fig. 3). Practically the only intestinal cancerous lesions to which the term "early" can be applied fairly have a polypoid character. The transitional forms with gross morphologic features other than those included under the term "polypoid" are rarely, if ever, demonstrated to us. This does not mean that all lesions classifiable as "polypoid lesions" are malignant or even potentially so. It does mean that such lesions may be cancer at the time, or definitely or potentially precancerous, and that there is no way short of careful microscopic examination of the entire lesion, to arrive at the status of certainty about their true nature. It follows (1) that extirpation is imperative every time such a lesion is encountered and (2) that the time to recognize intestinal cancer, if we are going to eradicate the lesion before metastatic spread has occurred, is at the stage when it is still a polypoid lesion and when it is a small lesion. I have indicated that we have the diagnostic implements with which to accomplish this.

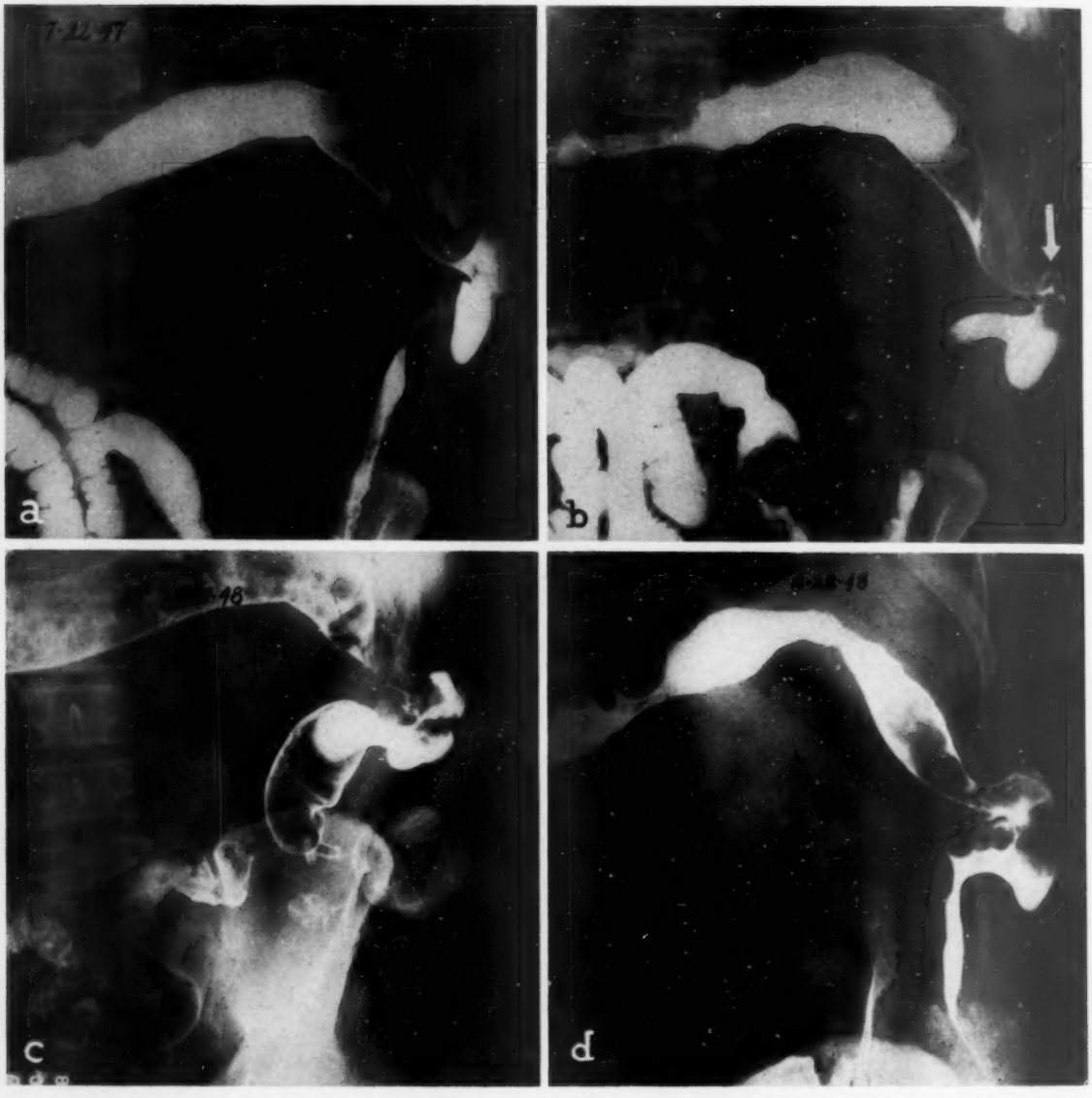

FIG. 3. The progression of a tiny polypoid lesion to an encircling, invading, frank carcinoma. The patient, a man fifty-five years of age, had an adenocarcinomatous polyp, grade 1, removed from the sigmoid colon by transcolonic excision in June, 1946. In November, 1946, a similar lesion was found in and removed from the upper descending portion of the colon by exteriorization of the affected loop and segmental resection. The exteriorized portion of intestine became involved in a ventral hernia. *a.* A small defect was found (arrow) which in July, 1947, was thought to be a residue of feces. *b.* (black arrow) In April, 1948, a large polypoid lesion was discovered in the sigmoid colon. This portion of the colon was resected and the lesion proved to be adenocarcinoma, grade 1. The significance of the deformity in the herniated loop (white arrow) was not fully appreciated at the time. *c* and *d.* Subsequent development of this lesion can be seen. When removed the lesion proved to be a polypoid adenocarcinoma, grade 1, penetrating the intestinal musculature but not involving the serosa.

It has been said that 50 per cent of the malignant neoplasms occurring in the rectum are discoverable with digital examination of the rectum. I assume, I think justly, that the lesions thus discovered are the larger lesions. Certainly a great majority of the small lesions will escape detection if rectal examination is considered complete when a digital examination has been done. It has been established that 60 per cent

of the malignant and potentially malignant lesions of the large intestine occur in the rectum and in that portion of the sigmoid colon which is within reach of the 25 cm. proctosigmoidoscope (Fig. 4). No proctosigmoidoscopic examiner who wishes to be called competent will refuse to accept the responsibility for the diagnosis of any neoplastic lesion in the rectum and lower portion of the sigmoid colon which has attained the macroscopic size of 5 mm. or more. Practically, this means that cancer in its curable stage can be recognized in the caudad 25 cm. or so of the intestinal tract invariably and with relative ease. Obviously then this examination is an unconditional requirement if the earliest possible recognition of intestinal cancer is to be accomplished, and it should be insisted on whenever there is even a remote suspicion of the presence of the disease. I have already suggested that the roentgenologic examination is no substitute for it. The roentgenologic examination may be considered the next best diagnostic maneuver, but I think we should be scrupulous enough to point out to consultant and patient alike the true nature of the situation. The patient certainly deserves to know that he has not had the best that modern medical diagnosis has to offer. Should the radiologist's consultant not appreciate this then I think he should be made aware of the facts of the matter, and in my opinion it is the responsibility of the radiologist to admit the limitations of his approach in this particular field.

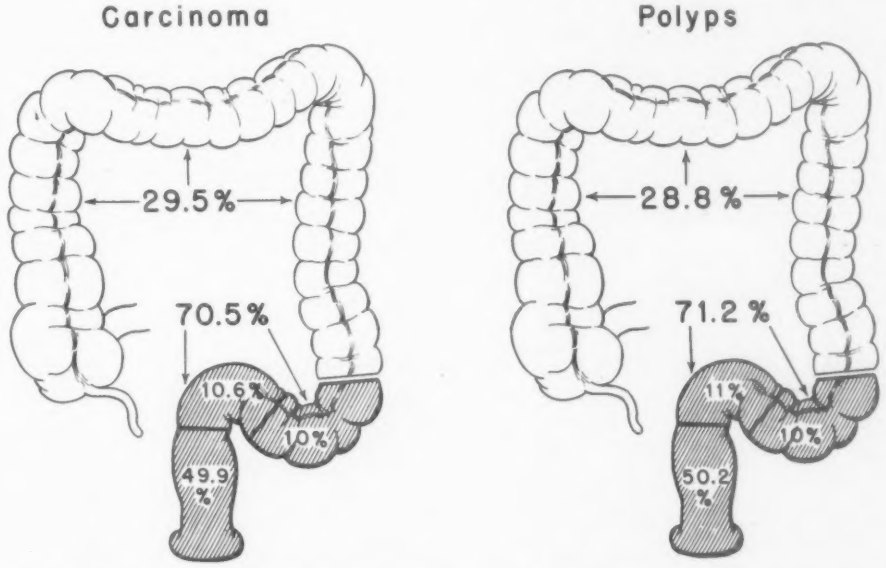

Fig. 4. The distribution of polyps and frank carcinoma in the various divisions of the large intestine. (From Buie, L. A., Smith, N. D., Jackman, R. L., and Hill, J. R. Fulguration of Polypoid Lesions of Terminal Portion of the Colon. An exhibit presented at a meeting of the American Medical Association, Chicago, Ill., June 21–25, 1948.)

Above the level of proctosigmoidoscopic reach where between 30 and 40 per cent of all intestinal neoplasms, malignant and benign, occur, the roentgenologic examination is the only diagnostic maneuver which offers reliable objective evidence about the existence of cancerous and potentially cancerous lesions. For practical purposes the lower limit of size of lesions discovered by roentgenologic examination may be said to be 1 cm. or slightly less (Fig. 5). Evidence of smaller lesions can, it is true, be elicited but it is frequently difficult to be certain at a single examination that we are not dealing with artefacts of one kind or another. I would be the last to maintain that the roentgenologic exploration of the mucous membrane of the large

FIG. 5. Tiny bean-shaped excrescence on the surface of the mucous membrane of the transverse colon. This was thought to represent a polyp, but it could not be demonstrated at subsequent examinations.

intestine is as definitive as that of endoscopy, but I am convinced that the roentgenologic examination of the large intestine can be made to elicit reliable evidence of small polypoid lesions without having a significant number of them escape detection. To accomplish this, roentgenologists must have the complete cooperation of their clinical and surgical consultants, especially in the preparation of patients for satisfactory examination. There must be an especially intimate cooperation between proctoscopist and roentgenologist. The lower portion of the sigmoidal and the rectosigmoidal regions are recognized as "dark regions" which offer difficulties for both examiners. It is then imperative that they deal with each other with complete frankness, soliciting mutual assistance, because it frequently happens that by dint of special effort one or the other can make his examination cover the region in an entirely satisfactory and secure manner.

I hope to have made a plausible case for the efficacy of the combination of proctosigmoidoscopic and roentgenologic examinations in the detection of early intestinal cancer. At this point the question "Who is to be submitted to these examinations and how often?" always arises. Theoretically, mass survey would be the answer. These examinations, however, are too cumbersome and involved to be readily adaptable to mass survey. Trained personnel is not available in nearly sufficient number to perform so many examinations, and the incidence of intestinal cancer is too low to make an undertaking of such magnitude economically feasible. Many of these examinations must be done on suspicion. Certainly it is imperative to submit any patient to most thorough intestinal investigation who has one or more of the following clinical symptoms and signs: (1) chronic loss of blood, manifested most frequently by the appearance of blood in the stools and less frequently by secondary anemia; (2) significant and persistent alteration in the functional activity of the intestine; (3) abdominal pain of a colicky, crampy type, often intermittent; (4) abdominal tumor. The patient with early or even late intestinal cancer may exhibit one, several, all or —of equal importance—none of these symptoms and signs, or others referable directly or even indirectly to the intestinal tract. Of the symptoms and signs enumerated only the appearance of blood in the stools and possibly the cramping, colicky pain, presumably caused by intussusception of a tumor can logically be considered as manifestations of early cancer. The tumor which is responsible for the other symptoms and signs enumerated will practically always prove to be a larger and a more advanced growth. The patient who disclaims intestinal difficulty, or minimizes the difficulty he may have been influenced to admit, probably deserves at least as much consideration as the one who is remarkably apprehensive about his intestinal activity or about his lack of it. For the present it seems practical only to

urge that physicians keep the problem of intestinal cancer in all of its aspects well in the foreground of consciousness, and hesitate not at all to advise complete intestinal investigation even on remote suspicion.

Since intestinal cancer, apparently more than any other form of internal cancer, yields to modern therapy with a low morbidity, a low mortality rate, and an encouraging survival rate if the disease is recognized at the stage before metastatic spread has taken place, we can well afford to display enthusiasm about the problem of intestinal cancer. I would urge those who are not radiologists or proctosigmoidoscopic examiners to resolve to give full cooperation to their roentgenologic and proctologic consultants to the end that maximal diagnostic yield may be derived from their special types of examination. Let radiologists and proctologists learn and apply with diligence, with devotion to patients' interests, and with hard work, the methods at their disposal, convinced that they are adequate, if competently applied, to solve the diagnostic aspect of the problem of intestinal cancer. The greatest problem is to determine the time and place to use these diagnostic implements, and to assist in training and encouraging more and more physicians to use them well.

To return, in conclusion, to the occasion of this address, I beg leave to quote briefly from the chapter on roentgenologic interpretation from the book which Carman and Miller[2] wrote more than thirty years ago. They seem to me to be very appropriate and complement some of the remarks I have made. They wrote: "The roentgenologic examination of the digestive tract is not a mysterious art requiring extraordinary talents. Neither is it a simple diagnostic method which can be learned in a day. Its successful employment demands industry, experience, judgment, and care, just as any other procedure in medicine—no more, but no less.

"The roentgenologist has to deal with shadows and shadow defects as signs of normal and pathologic conditions. These signs vary in frankness and degree. Some of them are . . . direct, showing as definite and permanent additions to or subtractions from the normal contour. Others are indirect, revealing only perversion of function, and the seat and nature of the pathologic change can only be inferred. . . . The exact worth of these signs in their fluctuating grades can be learned only by actual experience with adequate material and by following cases to the operating room and the post mortem table.

"Common sense has been defined as the ability to distinguish the important from the unimportant, and in this meaning every diagnostician has an abiding need of common sense.

" . . . Remember the rights of the patient. It is his just due that all features of his condition shall be properly weighed, not only separately but also in their relations to each other and as a whole. Somewhere and somehow the roentgenologic findings should be correlated with the clinical as well as all other data."

The urge to know the important, to elaborate roentgenologic symptoms and signs translatable into terms of pathologic anatomy, to cooperate and to consult, to modify opinion when not in agreement with fact and to keep the rights of the patient constantly in mind, are all features of the spirit of the man whose memory we are reviving this evening. May that spirit be kept alive in us and may we succeed in transmitting it to those to whom our own heritage will pass.

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SUBMUCOSAL GASTRIC LIPOMA

REPORT OF A CASE PRESENTING AN INTERESTING ROENTGEN SIGN

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THE infrequence of benign tumors of the stomach in relation to the countless roentgenographic examinations of the gastrointestinal tract is always a source of interest to radiologists who, as the vanguard in the search for early lesions of the stomach, are confronted with the problem of benignancy versus malignancy. Benign lesions are reportedly less than 5 per cent of all roentgenographically diagnosed gastric tumors. Of some fifteen different benign lesions of the stomach reported from various sources, lipomas constitute approximately 2 per cent.[1,3,4,5] In 1943, Hobbs and Cohen[2] in an excellent article summarized all previously reported cases, and added an unusual one of submucosal lipoma of the stomach causing intussusception. The case being reported was less unusual, but presented an interesting clinical story for over two decades, and was roentgenographically a definite problem.

CASE REPORT

The patient, a white male, aged sixty-nine, gave a history of epigastric distress for a period of twenty years. The character of discomfort was in the form of a dull, steady ache which persisted from two to ten hours at times, though unrelated to time of day or to meals or to type of food ingested. There was never any colic. Time interval between attacks varied from two months to two years. During episodes of gastric distress a proprietary medication would alleviate some of the discomfort. There was no weight loss, nausea or melena. Physical findings were non-contributory. In view of the clinical history, roentgen examination of the gastrointestinal tract was performed.

On roentgen study, a spherical polypoid filling defect was noted in the prepyloric portion of the stomach. This showed definite mobility within the lumen of the stomach for a distance of about 1½ inches. The overlying mucosa in all areas appeared intact. The surrounding wall was not fixed. Peristalsis would pass through this area and displace the polypoid defect, as if it were free within the gastric lumen. This behavior seemed to indicate that the lesion was pedunculated; however, careful search for a pedicle or its attachment was fruitless. Because of its intragastric motility, the final impression was that we were dealing with a pedunculated benign gastric polyp. We could not understand why the pedicle could not be demonstrated.

FIG. 1. Roentgenogram exhibiting the smooth oval prepyloric filling defect. On the original film mucosal folds across the tumor mass were very definite.

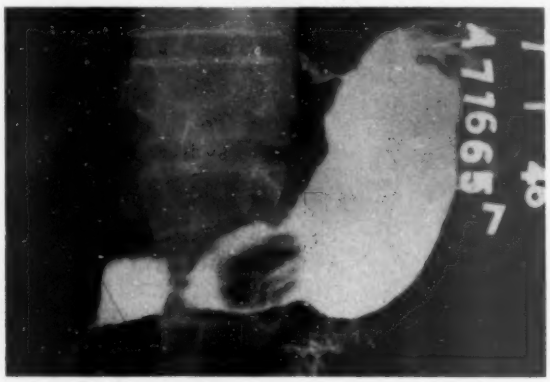

FIG. 2. Roentgenogram showing wave of peristalsis crossing the tumor site.

At surgery, on opening the peritoneal cavity a palpable mass was localized in the prepyloric portion of the stomach. There were no palpable nodes, and the liver appeared normal. A partial gastric resection was performed.

The pathologist reported a triangular fragment of gastric tissue measuring 7 by 10 cm., which contained a nodule 3.5 cm. in diameter elevating the mucosa 1.5 cm. above the surrounding level. It appeared to be fibro-fatty

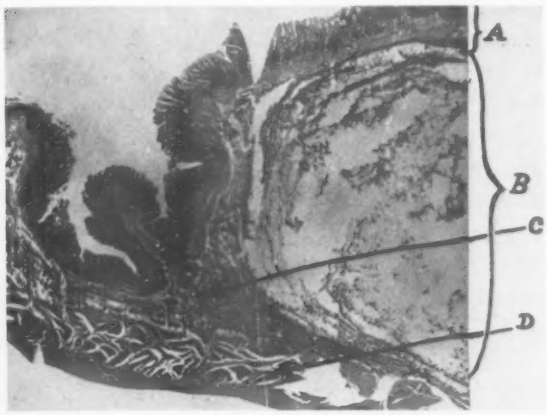

Fig. 3. Low power photomicrograph: *A*, mucosa; *B*, cross section through the lipoma, showing its submucosal location; *C*, submucosa; *D*, muscularis.

in consistency. The nodule was freely movable submucosally. Microscopically, a definite circumscribed lipoma of adult adipose tissue cells was found. The overlying mucosa was slightly compressed and showed plasma cells in the interglandular spaces. The remainder of the stomach was negative.

Comment. The above case has been presented because it exhibited a phenomenon which may prove helpful in establishing a diagnosis of submucosal lipoma of the gastrointestinal tract. This is free motion of a submucosal tumor. Such free motion may be erroneously interpreted as motion of an intraluminal pedunculated polyp, as was done in this case. However, if it is borne in mind that a movable tumor extending into the lumen of the gastrointestinal tract without a demonstrable pedicle may well be a submucosal lipoma, the diagnosis may be made in some instances.

CONCLUSION

A case of a submucosal lipoma in the prepyloric portion of the stomach has been presented which exhibited an interesting, and perhaps diagnostic, roentgen sign.

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INTRA-ABDOMINAL HERNIA*

DISCUSSION WITH REPORT OF A CASE OF INCARCERATION OF JEJUNUM INTO TRANSVERSE MESOCOLON

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HERNIATIONS which protrude into pouches or openings of the peritoneum rather than through defects in the retaining walls of the abdomen are variously designated as retroperitoneal, internal, or intra-abdominal hernias.

Such hernias are thought to be developmental in origin. From the early embryonic stage to the final adult arrangement, the abdominal contents undergo a series of complicated changes. During these stages in development certain fossae, peritoneal folds, and at least one opening later to become the foramen of Winslow are produced. Such changes are normal developmental phenomena. However, in the course of this complicated migration and rearrangement of the abdominal contents abnormal openings may occur, the result of faulty arrangement and development. The mobile organs of the abdominal cavity, especially the small intestine, are free to enter these abnormal orifices, and when this occurs an internal herniation is formed. Actually the finding of an intra-abdominal herniation is a rather unusual and rare condition. Such cases have been extensively reported by Treitz, one of the first to adequately describe the condition. Moynihan[8] has also treated this subject at length.

ANATOMICAL CONSIDERATIONS

The regions in which intra-abdominal hernias are prone to occur are chiefly (1) about the duodenojejunal junction, (2) the cecum, (3) the sigmoid mesentery, and (4) the foramen of Winslow.

The fossae into which the herniation of the bowel occurs may vary not only as to location and occurrence, but also as to size. When present, a fossa may be only a shallow dimple. Probably as such they are not too discernible, but, nevertheless, the possibility of their causing intermittent gastrointestinal disturbances must be considered. In addition, they are potentially the source of grave consequence, incomplete or complete intestinal obstruction resulting not too infrequently with fatal results.[13,14] Rarely these herniations may be so extensive as to accommodate the entire small intestine.[16]

Moynihan[8] has described nine such fossae, four of which are exceedingly rare. Two of these nine are the superior and inferior horns of the duodenal fossae of Treitz; the first occurring in approximately 40 per cent of autopsy material examined, the latter in 70–75 per cent. This is most constant of the peritoneal fossae considered. Its orifice opens upward and is located on the left side of the ascending arm of the duodenum at the level of the third lumbar vertebra. Another fossa, the paraduodenal fossa of Landzert, lies to the left and at a little distance from the ascending duodenum. This fossa results from the formation of a peritoneal fold by the inferior mesenteric vein.

Other openings about the mesentery include the mesentericoparietal fossa which is found in the proximal part of the mesojejunum behind the superior mesenteric artery and the mesocolic fossa which results from a fold containing the ascending branch of the left colic artery. As the gastrohepatic omentum is even more tenuous than the transverse mesocolon, it is not surprising that it is a route for re-entrance of the bowel into the general peritoneal

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cavity, and hernias which gain entrance to the lesser sac by the foramen of Winslow may leave it by this route.[2]

Adjacent to the cecum is the ileocolic fossa above the ileocecal valve. A similar fossa is found below the valve. Two other fossae, the retrocecal and the retro-appendicular are found in this region also. The intersigmoid fossa is reported as being present in 50–75 per cent of all cases. This lies between the root of the mesosigmoid and the parietal peritoneum.

SYMPTOMS

These are so obscure and few that rarely is a case diagnosed preoperatively or prior to necropsy.

Most cases give a vague history of minor digestive complaints and it is not until incarceration or strangulation of the bowel occurs with the manifestation of obstruction that the lesion is discovered. For this reason most of these cases are not diagnosed and come to surgery for an exploratory laparotomy for unknown cause of obstruction or for erroneously diagnosed conditions. Usually it is at this time only that the true nature of the lesion becomes evident. Study of the upper gastrointestinal tract by means of the barium meal is rarely done in these cases, probably due to the fear of converting a partial obstruction into a complete one. This may be an erroneous conception with too many proponents. It may be noted, however, that Case[1] has succeeded in the visualization of certain retroperitoneal hernias by roentgenographic study. In addition, most surgeons who may be anticipating a laparotomy dislike the finding of barium-filled bowel. As a result, most of these patients with obstructive phenomena are treated by the routine methods of decompression with the Miller-Abbott tube and Wangensteen suction. Roentgenographic examination is usually limited to the study of the abdomen for evidence of obstruction. The interpretation of a mechanical ileus is often made, but even with the findings of fluid levels within the distended small bowel loops it is usually difficult to make the true diagnosis. Perhaps if serial films were taken and showed persistent bowel loops with little change in position, a correct interpretation could be offered in a higher percentage of cases.

The physical findings in these cases are essentially those found in any case of obstruction. Occasionally when the hernia is about Treitz's fossa or through the foramen of Winslow, a fullness or sometimes a mass is described in the upper abdomen. If filled with gas this may be tympanitic on percussion. However, this should be considered as a most inconstant and not too reliable sign.

In lesions about the cecum and sigmoid, there may be fullness with tenderness in the right and left lower quadrants respectively. When on the right side a diagnosis of appendicitis is often made.[16]

CASE REPORT

E. J. was a male Negro coal stoker, aged fifty-two, who stated that he had been in usual good health when four hours before admission he was suddenly seized with severe cramping abdominal pain, diffuse in character. There was severe nausea and vomiting, but no hematemesis. He had a slight urgency to stool. He had had no previous similar attacks. He had had a normal bowel movement several hours prior to the onset of symptoms. Review of the other systems was essentially negative. Past history included pneumonia five years previously with no sequelae. He had had no previous abdominal operations. The family history was non-contributory.

On physical examination the patient was a muscular and well nourished middle-aged Negro male. He was of average height and weighed 180 pounds. He appeared acutely ill and was alert. Positive findings were chiefly limited to the abdomen, which was slightly distended, and there was a moderate diffuse tenderness and muscle spasm. This appeared to be most marked in the right lower quadrant. There were hyperactive peristaltic sounds. No mass or organs could be palpated. The external inguinal rings were intact and no cough impulse was felt. Rectal examination was negative.

The urinalysis and serological tests were negative. A complete blood count showed a normal red cell count and hemoglobin. There

FIG. 1. The barium-filled stomach approximately twenty minutes following roentgenoscopy. The incarcerated small intestinal loop may be defined projecting posteriorly and above the lesser curvature of the stomach.

was a leukocytosis with a predominance of polymorphonuclear cells.

A flat film of the abdomen taken within twenty-four hours of admission revealed some distention of small intestinal loops with several fluid levels. There were at least two distended loops of small intestine on the right lateral decubitus film. A roentgenographic interpretation of mechanical ileus was offered. The chest film was negative and no free air was defined beneath the dome of the diaphragm. Approximately forty-eight hours after the onset of symptoms following decompression measures, another flat film of the abdomen showed no distention of the small intestine and only a small amount of gas within the colon. A barium enema at this time revealed no roentgen evidence of disease.

The patient roentgenographically and clinically was well within forty-eight hours of his admission. Approximately one week after he became asymptomatic an upper gastrointestinal roentgen study was done. Following the ingestion of the barium meal the esophagus was normally visualized and emptied readily into the stomach, which was normal in size, shape, position, flexibility, mobility and peristalsis. The duodenal cap filled readily and showed no evidence of marked deformity or ulceration. There was no evidence of obstruction of the barium meal. Roentgenoscopically this patient was considered to be negative, but on subsequent follow-up films taken at hourly intervals, an abnormal loop of small intestine was observed just posterior to the stomach in position. This loop was reported as probably lying in the proximity of the ligament of Treitz (see Fig. 1 and 2). The barium was seen to be contained within this loop along with a small collection of gas for as long as four hours, and at the end of five hours a gas-containing loop could still be defined. The loop lay in an isolated and high position of the abdomen as compared with the remaining segments of the small intestine (see Fig. 3, *A* and *B*). It was felt that this point represented the site of partial obstruction which accounted for the patient's previous episode of obstructive symptoms. The roentgenographic interpretation of an internal hernia of the jejunum near the ligament of Treitz was offered.

In view of these findings and with consideration of the recent history of obstruction, it was felt that corrective surgery was indicated in the

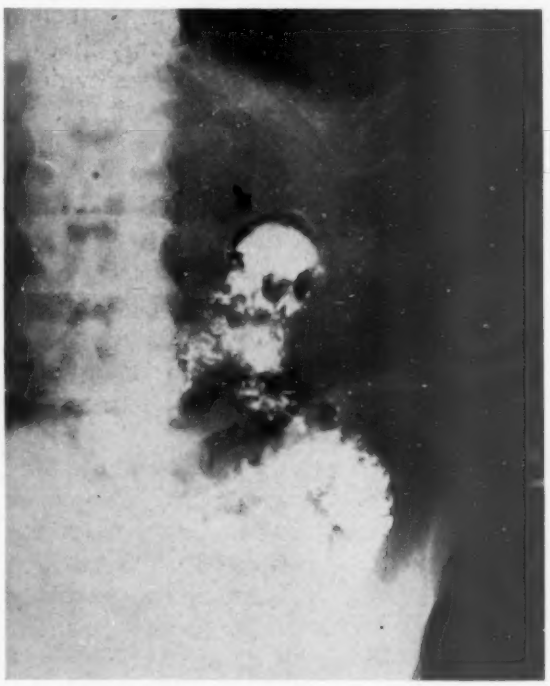

FIG. 2. Two hours following the ingestion of the barium meal. The stomach is empty and the isolated intestinal loop is seen to contain barium and air.

best interest of the patient regardless of the asymptomatic state of the patient at that time.

At operation there was an area of outpouching found in the antimesenteric border of the jejunum approximately 4.5 cm. distal to the ligament of Treitz. This outpouching measured about 3.0 cm. at its base and it was about 1.5 cm. deep. Its serosa and remaining jejunal wall appeared intact. It lay in a herniation of the transverse mesocolon in an avascular area just to the left of the ligament of Treitz. The outpouching of the transverse mesocolon corresponded in size to the outpouching of the jejunal wall. Exploration of the remaining abdominal organs revealed no gross pathological changes. In the repair the jejunal loop which was herniated was readily freed from its incarcerated position and the outpouching in the transverse mesocolon was closed. The large and small intestine were then replaced in their anatomical relationship. Recovery following the surgical procedure was uneventful. An examination six months following the operation revealed an asymptomatic patient who was able to continue with his work, which requires heavy labor.

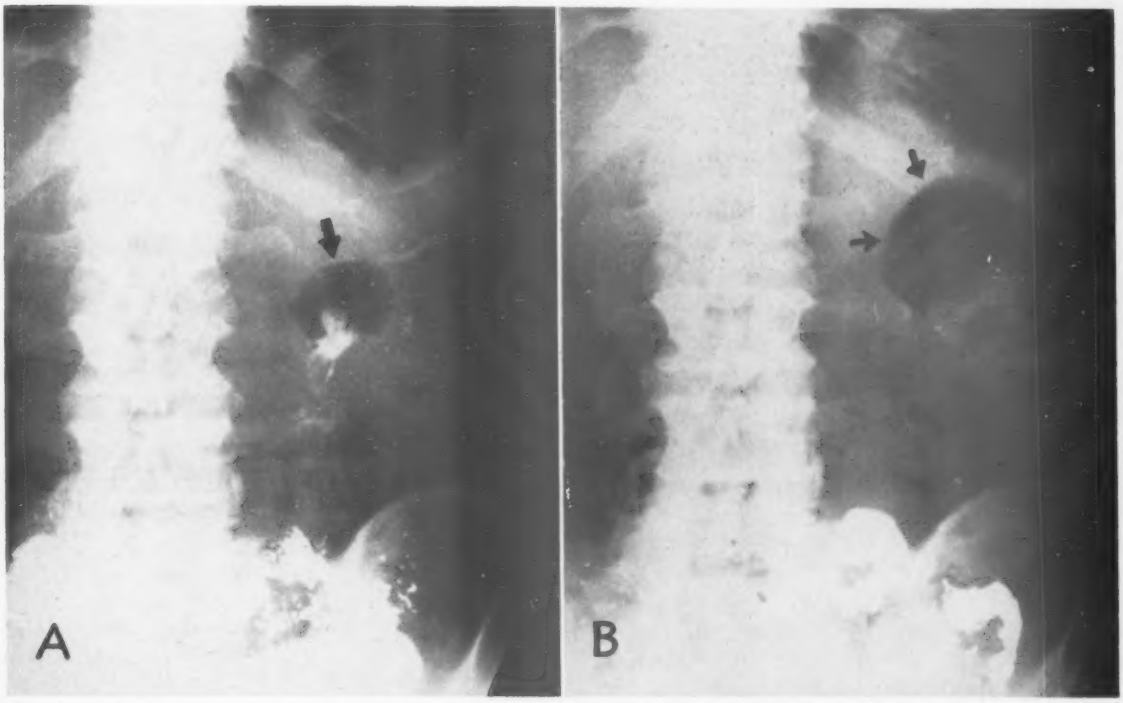

Fig. 3. The isolated air-containing jejunal loop three (*A*) and six (*B*) hours after the barium meal.

SUMMARY

1. A general review and discussion of the subject of intra-abdominal hernias has been given.

2. As it is usually difficult and unusual for a diagnosis of internal hernia to be made prior to surgery or necropsy, it is suggested that a more complete study in favorable cases by means of the barium meal might increase the incidence of correct diagnoses in cases presenting obstructive symptoms.

3. The finding of an isolated and distended loop of small intestine on the flat film examination of the abdomen is suggestive of a diagnosis of internal herniation in patients with partial obstructive signs and symptoms. If such a roentgenographic interpretation is inferred on the initial study of the abdomen, further investigation by means of a small amount of barium is probably indicated.

4. A report of a case of herniation of the

jejunum into the transverse mesocolon is presented.

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Vol. 64, No. 6

VISUALIZATION OF THE ABDOMINAL AORTA AND ITS BRANCHES FOLLOWING INTRAVENOUS INJECTION OF CONTRAST MEDIUM*

A REPORT OF FOUR CASES

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CONTRAST visualization of the abdominal aorta in the living by direct methods was first described by dos Santos[5] and his associates in 1929. These investigators initiated translumbar injection using various types of contrast media. Since then the diagnostic value of translumbar abdominal aortography has been established by other investigators.[2,3,4,6,7,9]

In 1939, Robb and Steinberg[8] published their method of angiocardiography by intravenous injection of 70 per cent diodrast. They reported opacification of the renal arteries in 2 instances.

Our interest in an intravenous method of opacifying the abdominal aorta and its branches was stimulated by the work of Weens *et al.*[10] who described the opacification of the renal parenchyma following rapid intravenous injection of contrast media.

PROCEDURE

With the patient supine on the roentgen-ray table an intravenous injection of 50 cc. (for adults) of 70 per cent diodrast is made according to the Robb-Steinberg technique. An initial 14 by 17 inch Bucky film is exposed at the estimated instant of abdominal aortic filling with the contrast medium. This interval is determined by the patient's circulation time, and is usually about ten seconds from the end of the injection. Three subsequent films are exposed at short intervals (four seconds or less). Initially we changed cassettes manually but now have constructed a simple five cassette (14 by 17 inch) tunnel, mounted with a Bucky diaphragm which is incorporated in the roentgen-ray circuit.

Using this procedure in a small group of patients, we have satisfactorily visualized the abdominal aorta, its major branches, and the opacified renal parenchyma. The following cases may serve to illustrate the value of this procedure in the diagnosis of diseases affecting the abdominal aorta and its branches.

CASE REPORTS

Case 1. A fifty-five year old cafe proprietor, female, was admitted on October 12, 1948, with paralysis of both lower extremities and incontinence of feces and urine. Two months before admission, she was injured when a truck crashed through the window of her cafe. At that time the extent of her injuries appeared to be only multiple contusions and lacerations. Two weeks later she developed sudden, excruciating pain in the lower back and lower extremities. Both legs rapidly became paralyzed and cold. Examination at that time by a private physician revealed absence of arterial pulsations in both lower extremities, glycosuria, and an elevated blood sugar. She was admitted to another hospital two days later and was found to be in mild acidosis. Pulsations were still absent in the arteries of the lower extremities, the feet and legs were cold to touch, and tendon reflexes were absent in the legs. There was complete anesthesia to a point just above the knees, and only vibratory, positional sense, and ability to distinguish between hot and cold remained below the knees. Intravenous pyelograms disclosed a non-functioning left kidney. She was treated for diabetic acidosis and rapidly improved. The arterial pulsations slowly returned to her lower extremities and the anesthesia of her lower extremities gradually lessened. At the time of admission to this hospital, both legs were warm and the arterial pulsations of both lower extremities were normal to palpation. Intravenous aortogram was

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performed on October 8, 1948, and the aortogram showed a normal appearance of the aorta, normal opacification of the right renal artery, but absence of opacification of the left renal artery (Fig. 1). The patient gradually improved and was discharged with slight foot drop and non-functioning left kidney, January 15, 1949.

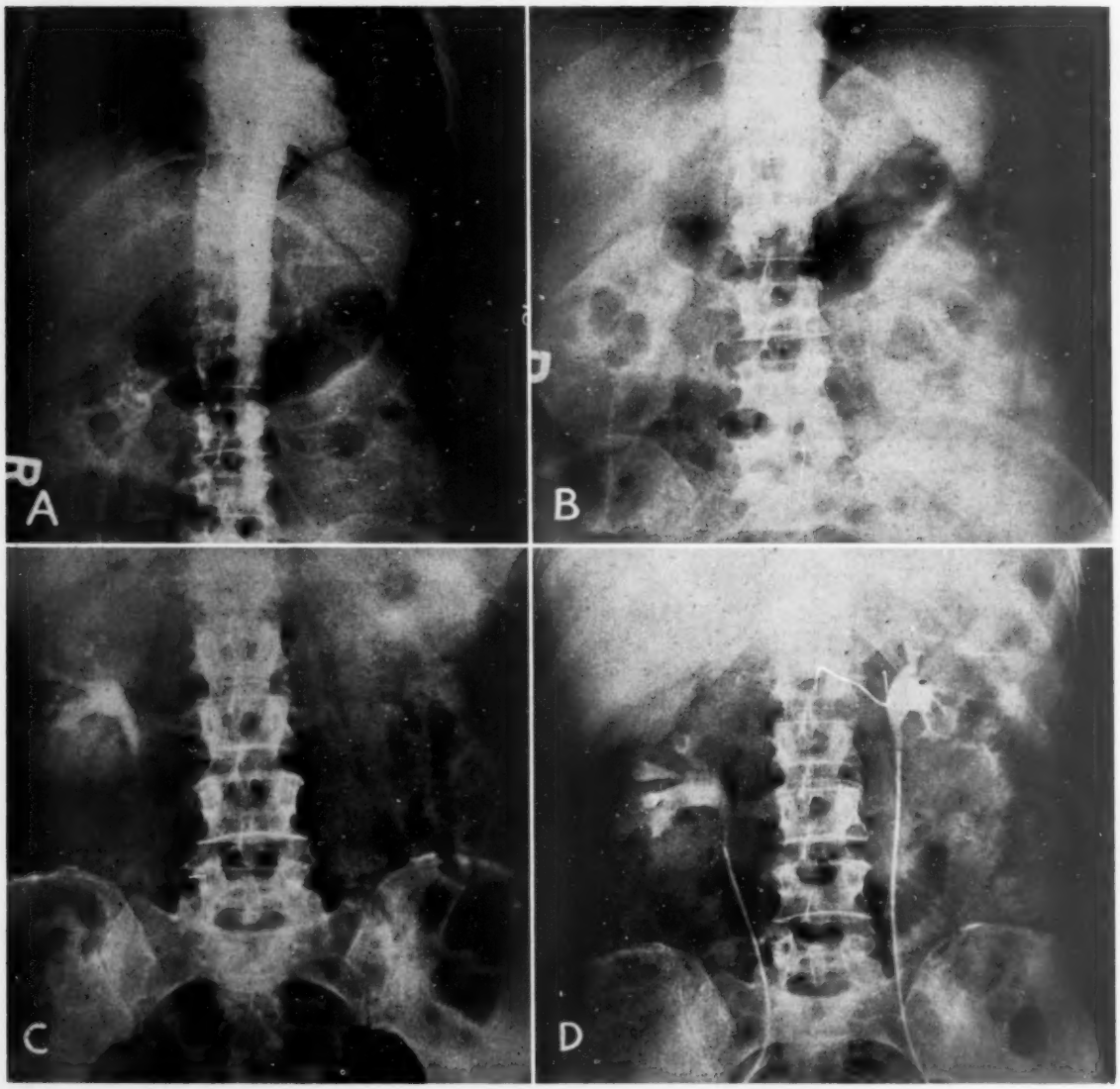

FIG. 1. Case 1. (*A*) The entire thoracic and abdominal portions of the aorta, and the proximal iliac arteries are filled with the contrast medium. The splenic artery and some of its branches are seen in the left upper quadrant of the abdomen. The hepatic artery is filled in its more proximal portions. The right renal artery and its branches are visualized. The left renal artery is not opacified. Branches of both the superior and inferior mesenteric arteries are opacified.

(*B*) Second film of series. The parenchyma of the right kidney is now opacified. The left kidney did not opacify. The contrast visualization of the stomach is caused by air in its lumen and opacification of its wall. The abdominal aorta can no longer be identified.

(*C*) An abdominal film thirty minutes after the aortogram. The calyces, pelvis, and upper ureter on the right are normal. There is no excretion of the contrast medium from the left kidney.

(*D*) Retrograde pyelogram. The soft tissue shadow of the left kidney is small in size. The middle and inferior calyces are slightly clubbed and small. The superior calyx and the extrarenal pelvis are normal in size. The soft tissue shadow of the right kidney is large. The calyces and pelvis are normal.

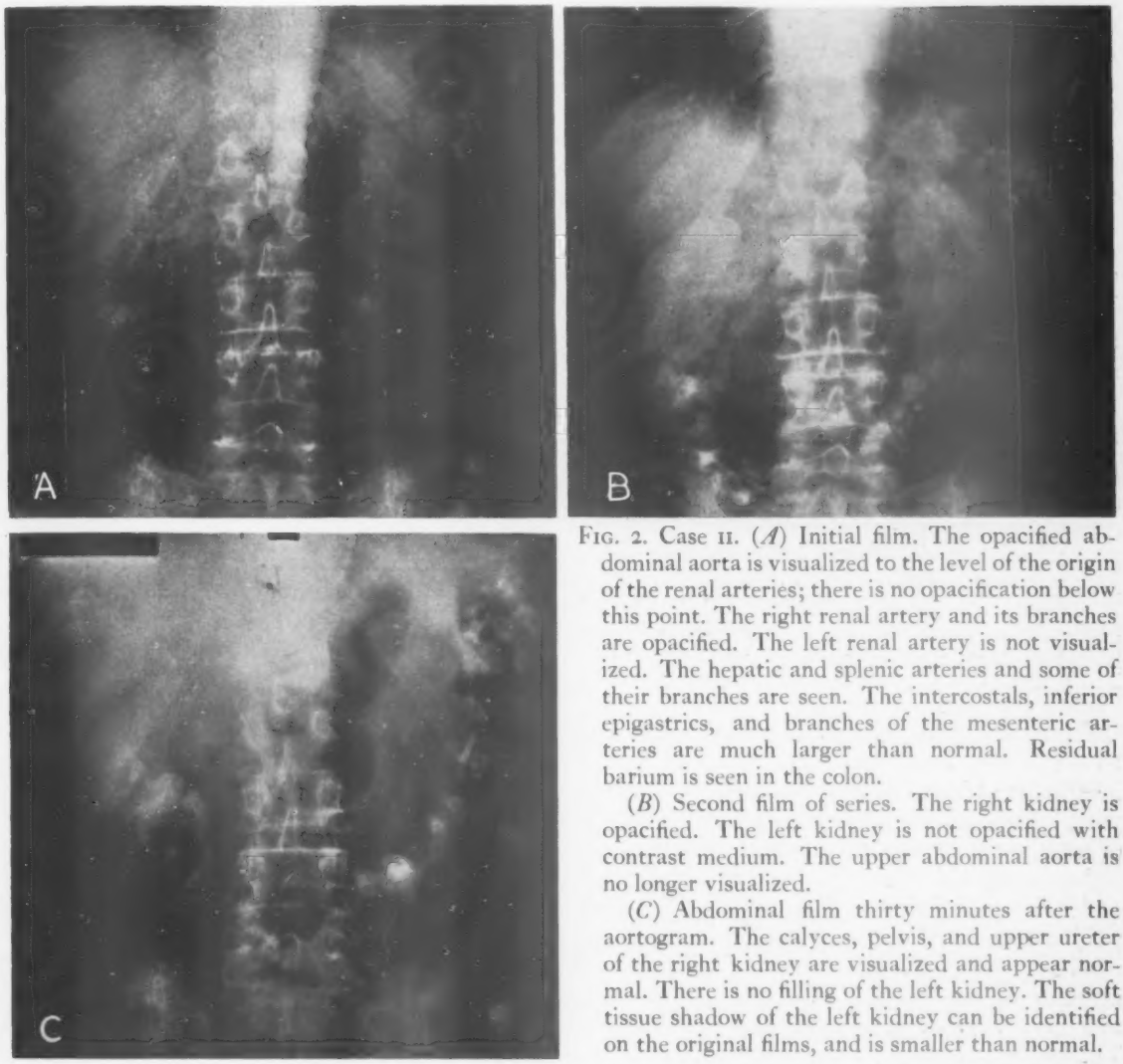

Fig. 2. Case II. (*A*) Initial film. The opacified abdominal aorta is visualized to the level of the origin of the renal arteries; there is no opacification below this point. The right renal artery and its branches are opacified. The left renal artery is not visualized. The hepatic and splenic arteries and some of their branches are seen. The intercostals, inferior epigastrics, and branches of the mesenteric arteries are much larger than normal. Residual barium is seen in the colon.

(*B*) Second film of series. The right kidney is opacified. The left kidney is not opacified with contrast medium. The upper abdominal aorta is no longer visualized.

(*C*) Abdominal film thirty minutes after the aortogram. The calyces, pelvis, and upper ureter of the right kidney are visualized and appear normal. There is no filling of the left kidney. The soft tissue shadow of the left kidney can be identified on the original films, and is smaller than normal.

Comment. The intravenous aortogram established the presence of an obstruction in the left renal artery which resulted in the absence of a nephrographic effect in the left kidney. This obstruction was believed to be a thrombus in the left renal artery.

Case II. F. D., a white male, aged fifty-eight, dated the onset of his present illness to ten years ago, when he had a sudden episode of numbness of his legs. This sensation was described as a transient, prickly feeling, without sequelae or residua. He was then well until 1943, seven years later, when he had an episode of weakness without loss of consciousness. Again, in May, 1948, he had a sudden episode of unconsciousness during hot weather and slumped over the steering wheel of his car. He regained consciousness and had no further difficulty. His legs tire easily and feel weak after prolonged walking.

Physical examination of the abdomen revealed pulsations of the abdominal aorta but absence of pulsations in the iliac arteries. The dorsalis pedis, posterior tibial, and popliteal arterial pulsations were absent bilaterally. Faint pulsations could be felt in the femoral arteries, the right being somewhat stronger than the left. Pulsations in the arteries of the upper extremities were entirely normal.

A plain film of the abdomen showed a small left renal shadow and a large shadow of the right kidney. An intravenous pyelogram revealed no excretion on the left side, but a normally functioning right kidney. The intravenous aortogram showed a lack of filling of

the aorta below the level of the origin of the renal arteries (Fig. 2). The left renal artery and parenchyma of the left kidney did not opacify. The right renal artery was opacified and a nephrogram was obtained on the right side. Many of the smaller abdominal arteries were tortuous and dilated.

Comment. Contrast visualization of the obstructed aorta was extremely important in this case because it determined the level of the lesion and demonstrated a well developed collateral circulation. The obstruction was in the region of the origin of the renal arteries, and because of its location, surgery on the aorta was not considered. It was felt that the possibility of damaging the normally functioning right renal artery far outweighed any advantages a reparative process would offer. Bilateral lumbar sympathectomies were recommended but, at the present writing, have not been performed.

CASE III. A physician, aged fifty-six, was admitted on April 2, 1948, with the chief complaint of intermittent claudication of three years' duration. Examination revealed absence of arterial pulsations in both lower extremities. Pulsations could be felt in the abdominal aorta as low as 3 cm. above the umbilicus. The palpable arteries of the upper extremities were markedly sclerotic. There was no evidence of atrophy of the muscles of the thigh or legs, and skin surface temperatures were normal. The clinical impression was thrombosis of the terminal aorta.

An abdominal film revealed marked calcification of the lower abdominal aorta and iliac arteries. An intravenous pyelogram disclosed excretion of the contrast medium by both kidneys, and a normal appearance of the calyces, pelves, ureters, and bladder. On intravenous abdominal aortography, satisfactory opacification of the aorta to a level just below the renal arteries was demonstrated.

Comment. On the basis of the clinical and roentgen findings in this patient, the diagnosis of thrombosis of the aorta was made. Because of the marked changes in the patient's peripheral vessels, it was believed that excision of the thrombosed segment of the aorta would not be feasible and that the benefit from sympathectomy would be extremely problematical.

CASE IV. This physician's wife, aged sixty-eight, was admitted on November 3, 1948, because of an episode of sudden severe pain in the left lower quadrant ten days before. The pain was relieved only by opiates hypodermically, and was accompanied by nausea and vomiting. This episode lasted only a few hours but there remained soreness and tenderness to deep palpation in the left lower quadrant. The physical examination revealed a questionable mass in the left lower quadrant which seemed to pulsate.

Barium enema revealed "an anterior and medial position of the lower descending colon probably caused by a deep mass in the left lower quadrant." A retrograde pyelogram was normal. An aortogram by the intravenous method revealed good filling of the abdominal aorta and the iliac arteries and no abnormalities of these vessels were noted. Each kidney showed a normal nephrographic effect.

Comment. A ruptured abdominal aneurysm was suspected, but was disproved by intravenous aortography. The patient improved during her hospital stay. No definite diagnosis was made, and she was discharged for further follow-up by her family physician.

CONCLUSIONS

We have found that the above described method of opacifying the abdominal aorta and its branches is a relatively simple procedure which may reveal important findings. It is a procedure that can be performed in most roentgenologic departments. Our patients who showed lesions in the aorta tolerated the procedure well. The contraindications to intravenous abdominal aortography are the same as those for angiocardiography.

We have had no experience with translumbar aortography, a method which requires skill in puncturing the aorta and a general anesthetic. In the twenty years since its introduction, it still has not gained widespread use.

SUMMARY

An indirect method of contrast visualization of the abdominal aorta and its branches is described.

Four cases are described in which this procedure was of value in the diagnosis.

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ADDENDUM

Since the acceptance of this paper, Case II has come to postmortem. A thrombus was found in the abdominal aorta, obliterating the left renal artery.

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THE ROENTGENOLOGICAL ASPECT OF INFECTIOUS MONONUCLEOSIS*

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THE important role assigned to roentgenology in tuberculosis and tumor detection and in the differentiation of obscure pulmonary and mediastinal lesions obliges the roentgenologist to be familiar even with pathologic lesions which are more exactly diagnosed by other laboratory methods.

Infectious mononucleosis is commonly regarded as a benign disease occurring sporadically or epidemically with various complications under the clinical picture of pharyngitis or tonsillitis, accompanied, as a rule by local or general lymph node involvement. Laboratory and blood tests are frequently omitted and a roentgen examination appears unnecessary.

During the last few years, the great diversity in the clinical appearance of infectious mononucleosis has been recognized,[6,8,12] and eventually these patients reach the roentgenologist with a tentative diagnosis of a pneumonitis, influenza, hepatitis or stomach ailment or of a pyelonephritis, leukemia, appendicitis, malaria, brucellosis or typhoid fever. All these erroneous diagnoses have been made in the past by competent clinicians in atypical cases of infectious mononucleosis, where adenopathy and sore throat were not present and the course of illness was obscure and protracted. These various diagnoses might well confuse the roentgenologist not familiar with the variability of the disease, for he sees only part of the picture and never the whole image. It is even conceivable that he might be called upon to give roentgen treatment to such a patient with

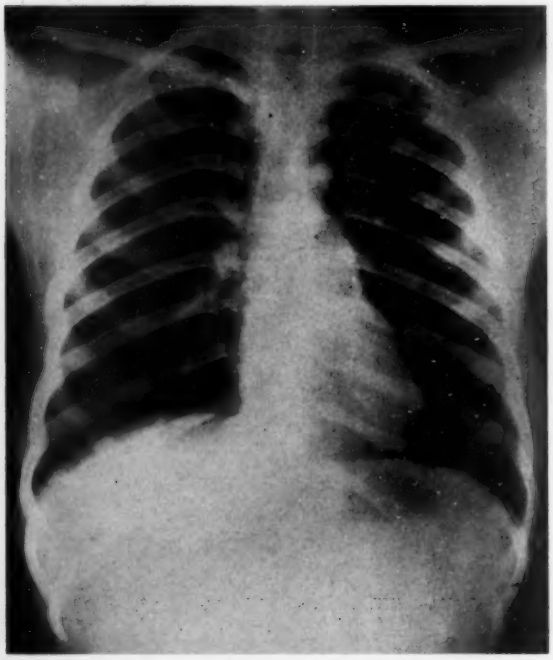

FIG. 1. Enlarged tracheobronchial and bronchopulmonary lymph nodes found in infectious mononucleosis.

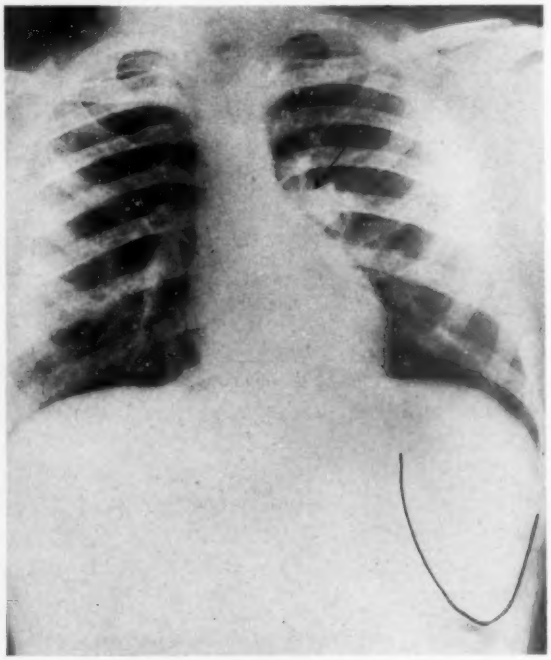

FIG. 2. Large hilar infiltration of the left hilus simulating a tuberculous infiltration, but due to infectious mononucleosis.

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lymph node involvement, under the diagnosis of leukemia or lymphoblastoma. The roentgenological literature on this subject is extremely scarce, and the condition we believe has not attracted the attention it deserves as a common differential diagnostic possibility.

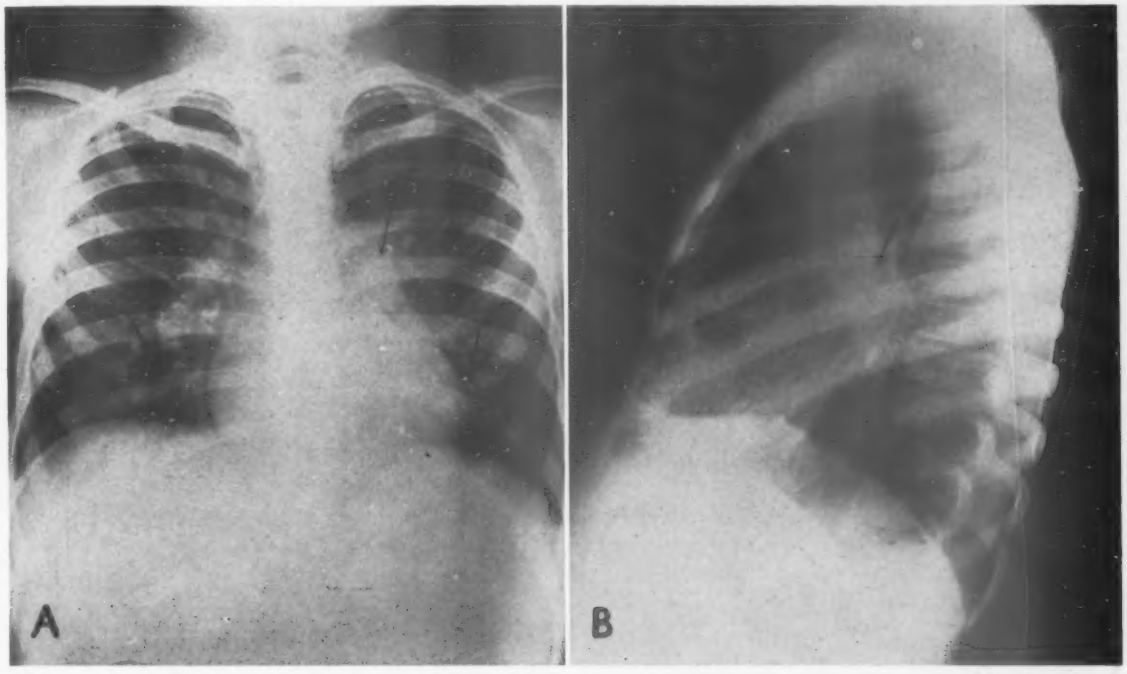

FIG. 3. (*A*) Marked swelling of tracheobronchial and bronchopulmonary glands leading to marked prominence of the pulmonary artery. Such glandular enlargement simulates a congenital lesion of the heart. The enlarged glands and pneumonitis in this case are due to infectious mononucleosis. (*B*) Lateral view of the same case.

The roentgenological findings consist first in the detection of the visibly enlarged hilar lymph nodes (Fig. 1 and 2). The appearance of such deep lymph nodes may not be accompanied by external adenopathy. It is now proved, as had been postulated by some authors[3] that the glandular involvement may be confined to deep-seated glands: mediastinal and mesenteric glands. More commonly they are part of the general adenopathy, and may only precede the palpatory evidence of glandular swelling. Such glands may persist, as we observed in one case, for months and even years, and become a roentgenological enigma.

These mediastinal lymph nodes are of sufficient size to be visible through and beyond the vascular hilar shadow. They are discernible on the lateral view as well as on the posteroanterior view. In 4 of the cases which we observed they were close enough to the pulmonary vessels and the waist of the heart to simulate a congenital heart lesion (Fig. 3, 4 and 5), particularly a patent ductus arteriosus Botalli; yet not one of these cases presented a congenital heart lesion. In none of the cases which we observed did the glands form masses suggestive of lymphoblastoma. We observed emanating from the hilus fine strands of densities commonly but not too accurately described as increased bronchovascular markings (Fig. 6 and 7). They were in some cases intense enough and surrounded by spotty densities to be interpreted as atypical pneumonia. The hematological and serological tests proved them to be cases of infectious mononucleosis. It is very possible, however, that the same unknown agent which causes infec-

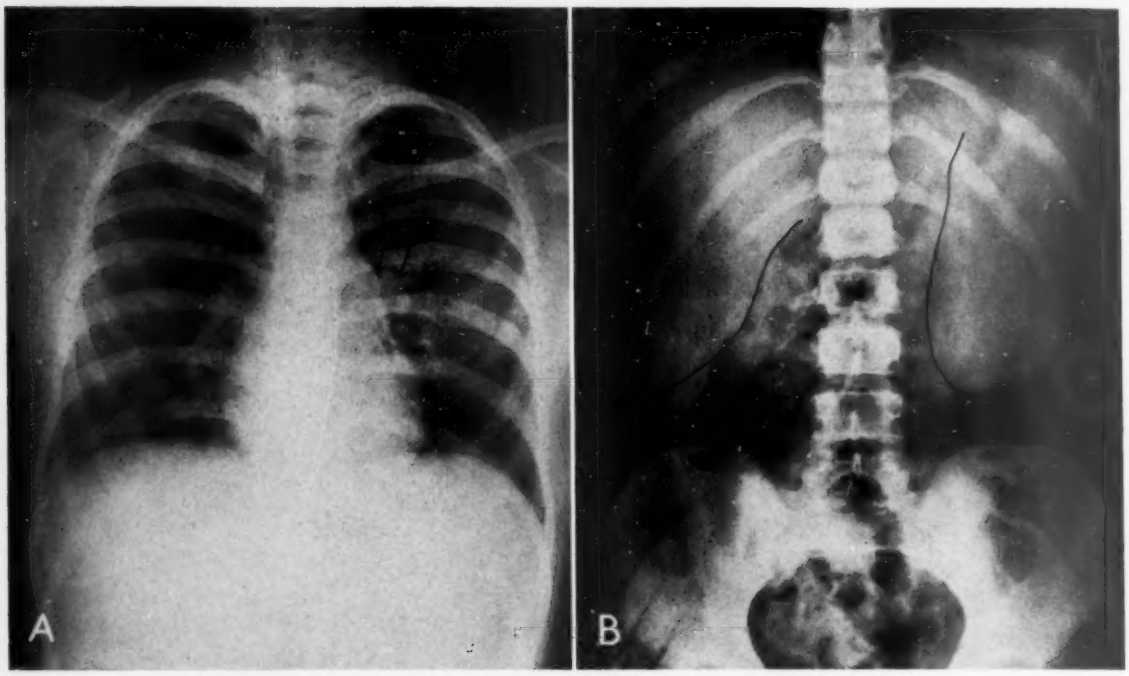

FIG. 4. Case 1. (*A*) Large left-sided hilar glands causing prominence of the pulmonary cone and artery. Spleen and liver are enlarged. Infectious mononucleosis in the disguise of a subacute abdominal condition. See text. (*B*) Flat film of the abdomen, same case.

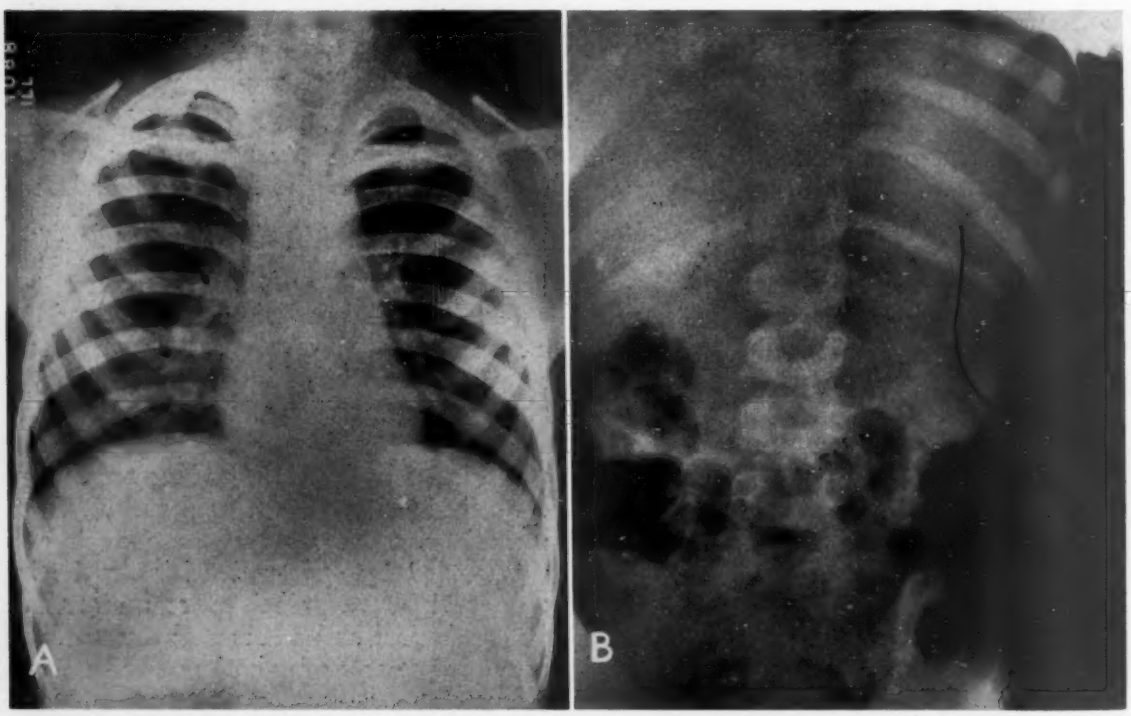

FIG. 5. (*A*) Atypical pneumonia with enlarged hilar gland. Infectious mononucleosis. Discharged after four days as markedly improved, with normal temperature. (*B*) Spleen considerably enlarged.

tious mononucleosis might cause an atypical or virus pneumonia. Wechsler *et al.*[17] reporting an epidemic of infectious mononucleosis in an Army post found 14 out of 556 cases with roentgen evidence of pneumonitis. In a fatal case described by Ziegler[18] the autopsy revealed histopathological findings consisting of interstitial and perivascular foci of mononuclear cells scattered throughout the lungs. In a case reported by Rodstein,[14] poorly circumscribed patches of density were observed on the roentgenogram over both lower lobes, suggesting the broncholobular and bronchopneumonic involvement of a primary atypical pneumonia. In another case, reported by Hirschboeck,[9] the film taken on the seventh day after admission indicated a bilateral bronchopneumonia. Five days later the roentgenologist suggested miliary tuberculosis as the probable diagnosis. Three weeks later the lung cleared completely and the case proved to be one more variation of the multifacetted appearance of infectious mononucleosis.

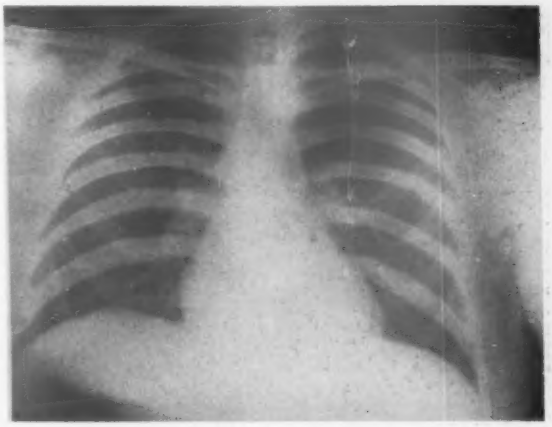

FIG. 6. Bedside film. Male, aged thirty-three. Fever and chills for one week, no response to penicillin and sulfa drugs. Roentgenological picture of virus pneumonia. Heterophile antibody test clears the case as one of infectious mononucleosis with lung manifestations.

The third roentgenological feature is the enlargement of the spleen. The roentgenogram is of decided value in the estimation of spleen size (Fig. 6*B*). Contratto,[3] reporting on 196 cases of infectious mononucleosis, found a *palpable* spleen in 91

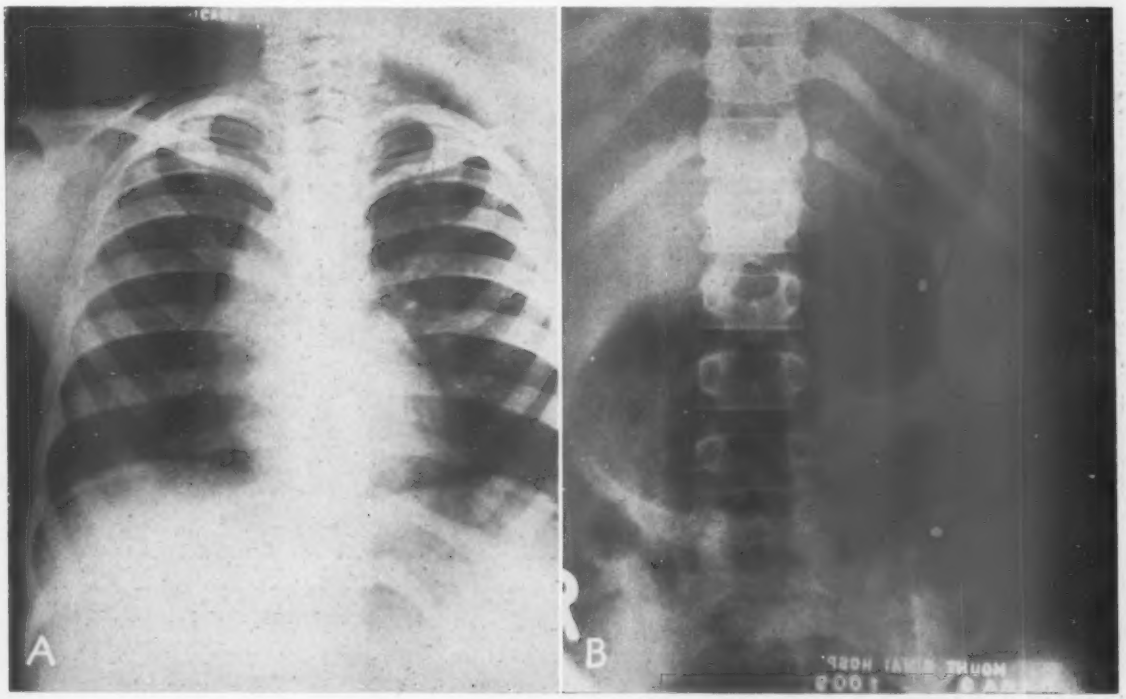

FIG. 7. (*A*) and (*B*) A seven year old girl with temperature up to 103° F., cough and sore throat. No response to penicillin. Roentgenological findings: Paramediastinal glands enlarged on the right side. Enlarged spleen. Diagnosis: Infectious mononucleosis.

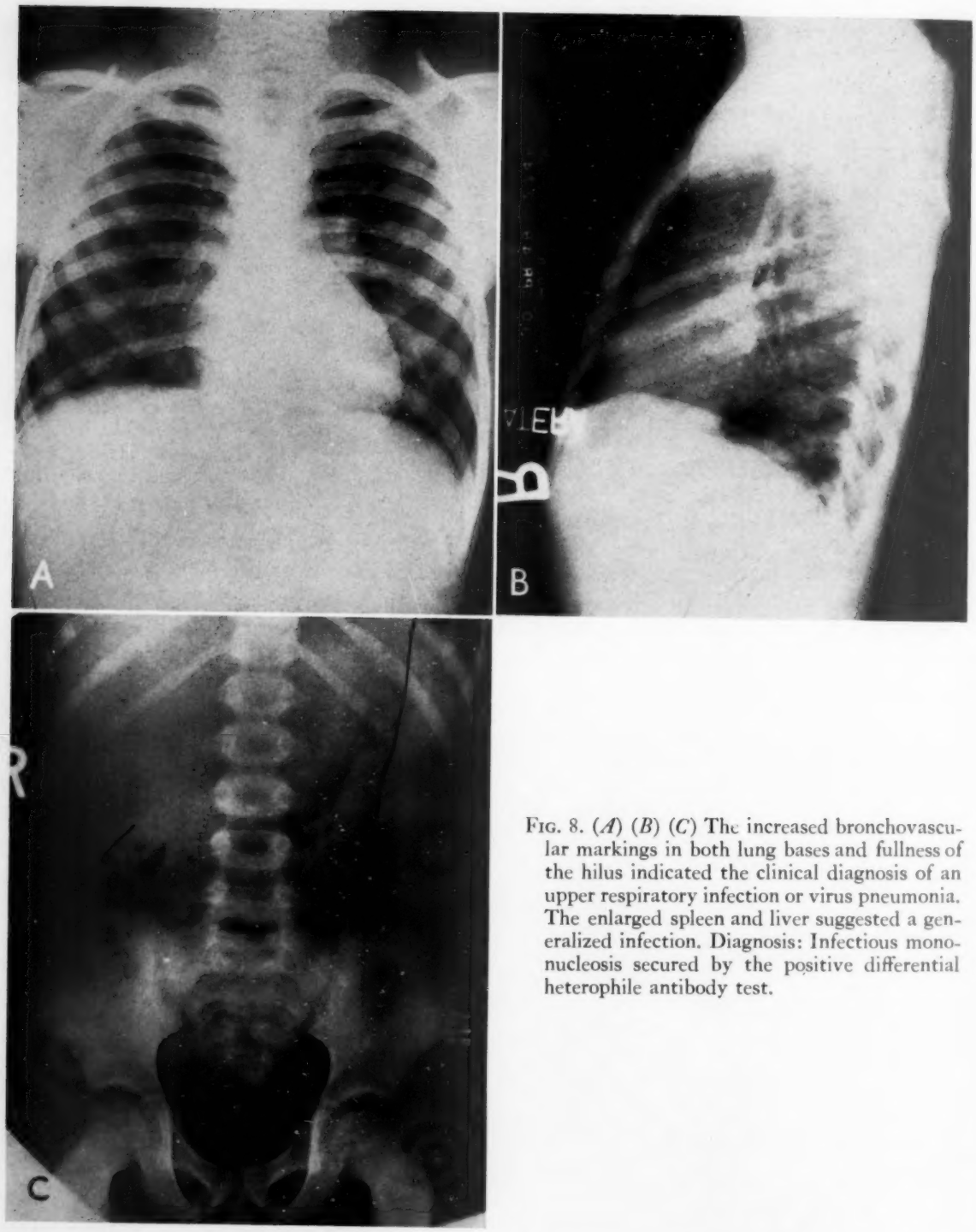

Fig. 8. (*A*) (*B*) (*C*) The increased bronchovascular markings in both lung bases and fullness of the hilus indicated the clinical diagnosis of an upper respiratory infection or virus pneumonia. The enlarged spleen and liver suggested a generalized infection. Diagnosis: Infectious mononucleosis secured by the positive differential heterophile antibody test.

patients, but 67 of these patients had spleens which could barely be felt, and only 15 where the spleen was easily palpable. Our own observation showed a high percentage of visible spleen enlargement and a degree of enlargement out of proportion to the lymphadenopathy. The spleen in infectious mononucleosis is soft and not as resistant as in leukemia and Hodgkin's disease. Palpation is not too

reliable and may even be dangerous. Rupture of the spleen was reported in at least 13 cases, and the roentgenologist who is called upon to make a colon examination in such a case has to be particularly careful as even the exertion of a bowel movement has caused a rupture of the weakened capsule of the spleen. On a simple flat film we have frequently demonstrated spleens of 18 cm. in length and a considerable increase in width. Such spleen enlargement might remain for months and even years; in one case such enlargement remained even seven years after recovery. This differential diagnostic possibility should be kept in mind.

In cases complicated by *hepatitis* the splenomegaly was frequently accompanied by an enlargement of the liver. In 3 of our cases the liver was slightly enlarged and clinically palpable. One of these had a gallbladder examination and showed nonvisualization of the gallbladder. Hepatitis is present with or without jaundice in a high percentage of cases and has been previously ascribed to cholecystitis and common duct obstruction by enlarged lymph nodes; yet it is more recently recognized as a type of infectious hepatitis. Such hepatitis, usually mild, but at times severe and frequently the cause of delayed recovery, might appear as cholecystitis or as a gastrointestinal disorder. The findings of concomitant gastritis and duodenitis in roentgenograms, and of an irritable and spastic colon with diarrhea, are common to all types of hepatitis. Enlargement and dysfunction of the liver might persist for weeks and months and might during this time be the cause of a grave diagnostic error. Certain obscure cases of moderate hepatomegaly in young persons with or without jaundice which failed to eliminate gallbladder dye might be due to the hepatitis accompanying infectious mononucleosis.

An acute abdominal condition has been simulated by infectious mononucleosis as in the case reported by Davidsohn.[4] Appendicitis and inguinal hernia were other clinical impressions which were spared surgical intervention only by the prompt recognition of the underlying disease. In one of our recent cases, the history was that of a stomach ailment with epigastric pain relieved by food intake. There were nausea, vomiting and regurgitation of solid food. The symptoms persisted for years. Three to four days before admission the patient suffered an acute exacerbation and it was thought likely that he had a recurrent ulcer. Yet roentgenograms revealed no sign of an ulcer, either recent or old; instead an enlarged spleen was found. It was only through the blood picture and the differential antibody test that infectious mononucleosis was recognized here in its disguise of an old recurrent ulcer. Direct and indirect evidence of mesenteric adenitis will be more frequently found, as our awareness of the condition grows.

An abnormal heart shape might occur with infectious mononucleosis due to acute myocarditis. Electrocardiographic changes are reported by Wechsler *et al.*[17] in 53 out of 233 patients. In one case of Bradshaw a mitral stenosis followed an attack of infectious mononucleosis. Numerous instances of transitory or permanent damage to the central nervous system, cases of psychosis and paralysis of the respiratory tract, encephalomyelitis and oculomotor disturbances are known; however, we have had no occasion to observe them—they rarely present a roentgenological problem.

The diagnosis can be established only by the laboratory:

1. On the basis of the hematological picture with morphological changes: the presence of the "atypical" lymphocytes and the high percentage of such lymphocytes.

2. On the basis of the heterophile antibody test. In each case the "presumptive"[11] test should be complemented by the "differential" test developed by Davidsohn.[5,6] This test, if positive, specifically indicates infectious mononucleosis. Yet none of these tests is positive in each case and the tests are frequently negative during the first week and may become positive as late as

the seventh and sixteenth day of the infection. It is particularly during this period that a roentgen examination is requested and we can be instrumental in suggesting the diagnosis on the basis of spleen enlargement, the enlargement of the hilar glands and an atypical pneumonitis.

CASE REPORTS

CASE I. Infectious mononucleosis resembling an abdominal condition or urinary tract infection (Fig. 4, *A* and *B*).

B. F., a ten year old white female, has had a burning sensation on urination for the past three to five months. In addition, she had enuresis unless awakened during the night. Onset of present illness was about two weeks ago when she complained of general abdominal distress. Fever began five days ago and went up to 103° F. No response to sulfa drugs or penicillin. She developed a dry cough, but no sore throat. The breath sounds in the right base were suppressed. No rigidity of the neck was present but many large cervical lymph nodes could be felt. The spleen reached 2 to 3 cm. below the rib margin.

Roentgen Findings. The roentgenogram showed large hilar lymph nodes in the left hilus with marked prominence of the pulmonary cone and artery imitating a congenital heart lesion, more particularly a ductus Botalli. Enlarged lymph nodes were also seen on the right side and on the lateral view in the hilar region. The spleen was markedly enlarged, measuring 19 cm. from the upper to the lower pole, 7.5 cm. in transverse diameter. The liver also appeared proportionately enlarged, its lower margin being localized at the level of the fourth lumbar vertebra, but flared upward in a receding line. Its longitudinal diameter was about 19 cm. at its outer edge. *Summary*: Visibly enlarged hilar lymph nodes are seen on the anteroposterior and lateral views simulating a congenital heart lesion. Markedly enlarged spleen; moderately enlarged liver.

The laboratory report showed a red blood cell count of 4,290,000, a white blood cell count of 14,500; hemoglobin 13.5 gm.; 85.5 per cent. The differential count: stab forms, 2 per cent; segmented, 30 per cent; small lymphocytes, 73 per cent, large lymphocytes, 12 per cent. Many atypical lymphocytes[1] were seen. The heterophile antibody tests, presumptive and specific (Davidsohn test), were positive for infectious mononucleosis. The child was discharged on the fourth day as on the way to recovery.

CASE II. Infectious mononucleosis with hepatitis presenting abdominal complaints and lumbar pain.

E. K., a white female, aged twenty-three, was admitted to Mount Sinai Hospital on December 14, 1948. The patient had apparently been in good health until the afternoon prior to admission when she developed a dull aching feeling in the lumbar area with occasional radiation of pain to the right upper quadrant of the abdomen. No other symptoms, and no antecedent upper respiratory infection.

The morning of admission the pain became more noticeable and radiated frequently to the right upper quadrant of the abdomen. Movements and postural changes aggravated the pain. The afternoon of admission the pain became more constant, gnawing in character and associated with malaise, chilliness and sensation of fever.

Physical examination revealed a twenty-three year old white female, not acutely ill. There was no adenopathy. The liver was palpated one to two fingerbreadths below the costal margin and was slightly tender.

Roentgen Findings. Film of the abdomen essentially negative. The spleen is not enlarged.

Laboratory reports were essentially negative at first except for traces to two plus blood found in the stool. Initial liver function tests were within normal limits except for three plus cephalin flocculation and 7.5 icterus index. Heterophile agglutination test for infectious mononucleosis was negative on the third hospital day and again on the seventh hospital day.

CASE III Infectious mononucleosis complicated by hepatitis with features of recurrent ulcer.

B. B. a white male, aged thirty, entered Mount Sinai Hospital on April 19, 1948. The patient had suffered from epigastric pain on occasions for the past six years. The pain began when patient was working as a bell-hop and eating his meals at irregular intervals, and was relieved by food. The patient also had nausea and vomiting after eating fried or fatty foods. Three to four days prior to admission the patient suffered an acute exacerbation of the above symptoms. He had been unable to

retain foods as well as before. The pain had been present almost constantly, and had not been relieved by food he had eaten and retained. Solid foods were regurgitated almost immediately after eating, and there was no relief of the pain following emesis. His bowel habits had not changed, but his urine had become darker.

Physical Examination. Well developed, well nourished white male not acutely ill. Blood pressure 114/80, pulse 76, respirations 24. Posterior cervical adenopathy was noted, but no other adenopathy; spleen not palpable. Tenderness was present in the epigastrium and right upper quadrant and the liver was enlarged one fingerbreadth. No other abnormalities were noted.

Roentgen Findings. No evidence of an ulcer of the stomach or duodenum. There is a pressure defect on the outer curvature of the stomach caused by the enlarged spleen.

Laboratory reports showed the following: Urine, a trace of albumin. The icterus index was 18 units. The blood count was: red blood cells 4,860,000; hemoglobin 15 gm. 84 per cent; white blood cells, 12,000; with a differential count of 3 per cent stab forms, 12 per cent segmented, 1 per cent eosinophils, 1 per cent basophils, 6 per cent monocytes, 77 per cent lymphocytes. The lymphocytes appeared toxic. Heterophile-presumptive test was positive to 1:14, 336 and differential positive to 1:3, 584. The urine-urobilinogen was not increased, the prothrombin time was normal, the thymol 14.5, serum cholesterol 291 and esters 184, 4 plus cephalin flocculation, and the hemosulphthalein showed a 25 per cent retention. Course was uneventful and the patient was discharged on the ninth hospital day unimproved.

CONCLUSIONS

1. Infectious mononucleosis is a common widespread disease which will frequently come to the roentgenologist's attention in the form of a pharyngeal edema, a virus pneumonia, an appendicitis or cholecystitis, a leukemia or lymphoblastoma, malaria, brucellosis or colitis.

2. Attention is called to the frequent involvement of hilar glands sometimes without, or before peripheral lymph glands are palpable. Such hilar gland involvement accompanied by marked splenic enlargement permits the roentgenologist to suspect the diagnosis, which, however, can be established only by the characteristic blood picture and the presumptive and differential antibody test.

3. The lung markings are frequently increased and simulate in every respect the picture of an atypical virus pneumonia. These lung changes are due to perivascular and interstitial foci of atypical lymphocytes and in some cases to true intra-alveolar infiltration with mononuclear cells.

4. The enlargement of the spleen is evident roentgenologically and more reliably demonstrated by palpation and percussion of the spleen. The enlargement may persist for months and years after clinical recovery.

5. The frequency of hepatitis with or without jaundice in infectious mononucelosis may lead to roentgen examination of the stomach, in which case a hypertrophic gastritis will be found. Examination of the gallbladder may result in non-visualization of the gallbladder. An examination of the colon is particularly contraindicated. Mesenteric glands may give direct and indirect signs of their pathologic enlargement and thus contribute to the diagnosis of infectious mononucleosis.

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VOL. 64, NO. 6

INTESTINAL GAS PATTERNS AS A DIAGNOSTIC AID

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IN THE past thirty-five years the interpretation of the significance of gas in various segments of the gastrointestinal tract has become fairly well standardized. The roentgenological diagnosis in such cases depends upon the dilatation of the bowel and the accumulation of fluid and gas. In the erect position, the radiologist may often see fluid level lines surmounted by dome-shaped gas bubbles throughout the distended loops of intestine. Although the fluid level lines with their characteristic air caps cannot be seen in preliminary films taken in the horizontal position, nevertheless the dilated loops of intestine are readily noted

From the roentgenological point of view, when the small intestine is seen to be markedly distended with gas and fluid without any gas being present in the colon, that is considered to be presumptive evidence of a small intestinal obstruction. The absence of gas in the colon is considered to be of considerable diagnostic significance in a case of this type. It is assumed, of course, that the preliminary film had been taken before an enema was given. An enema might eliminate the colonic gas with the result that a correct evaluation of the roentgen evidence would not be obtainable. Most radiologists agree that when the obstruction of the small intestine is mechanical in origin little or no gas is demonstrable beyond the obstructed area. If the cause of the distention is paralytic, then gas may be seen throughout the bowel including the colon. The presence of gas in the colon would then suggest that the distention in the small intestine was due to such other causes as might produce paralytic ileus.

From the preceding agreement of radiological opinion it would follow that a markedly distended small intestine with a colon containing no gas constitutes the roentgenological diagnostic criterion for small intestinal obstruction. Similarly, an obstruction of the pelvic colon would be accompanied by a distention of the colon proximal to the point of obstruction. The small intestine may or may not present intestinal distention in such cases, depending upon the patency of the ileocecal sphincter. With a closed sphincter, a virtual closed loop obstruction of the colon may occur so that the large intestine would be tremendously distended with gas and very little distention noted in the small intestine. On the other hand, a slowly progressive obstruction of the left colon associated with a non-competent ileocecal valve presents a picture of marked distention of both the small and large intestine.

Jarre[1] is of the opinion that one should not depend too much upon the appearance of characteristic roentgen gas shadows. The most characteristic feature of such gas patterns is their changeability. It has often been called to our attention by Jarre that to depend entirely upon some standardized gas pattern in the diagnosis of types of intestinal obstruction is to invite disaster. The 2 cases presented in this paper demonstrate this concept to be valid.

In the first case, a patient is presented with a tremendous distention of the small intestine and no gas in the colon (Fig. 1). For this reason, a roentgen diagnosis of small intestinal mechanical obstruction was made and operation advised. In the second case, the preliminary film demonstrated a marked small intestinal distention with no gas in the colon (Fig. 3). A diagnosis of mechanical small intestinal obstruction was therefore made. The error of both diagnoses will be evident in the cases presented.

[1] Jarre, H. Personal communication.

FIG. 1. Case 1. Note the characteristic gas pattern of small intestinal distention. No colonic gas visible.

CASE REPORTS

CASE 1. S. B., a sixty-two year old white male, was admitted to the hospital on May 7, 1949, complaining of pain in the abdomen. He had been vomiting for the previous two days. He had had no bowel movement and his abdomen had become progressively more distended. Upon admission to the hospital his temperature was 98° F., pulse 70, and respirations 20. Upon examination, he was seen to be an elderly white man of about the stated age. He appeared to be acutely ill. The only relevant physical findings were confined to the abdomen. There was diffuse tenderness over the entire abdomen and marked distention. No peristaltic activity was audible by auscultation. No fluid wave or shifting dullness could be demonstrated. On rectal examination a doughy, very tender, ill defined mass was palpable in the pelvis.

A preliminary film taken May 8, 1949, showed marked distention of the small intestine with gas. A small amount of gas was noted in the cecum and none in the remainder of the colon. On this basis a roentgen diagnosis of a mechanical small intestinal obstruction was made (Fig. 1).

On the basis of the physical findings, a diagnosis of paralytic ileus due to pelvic abscess was made despite the roentgen diagnosis. A Cantor tube was passed and intestinal intubation started. Intravenous alimentation was instituted to restore the altered fluid and electrolyte balance, and penicillin and streptomycin were given.

On May 9, 1949, a preliminary film was again taken with the report as follows: An intestinal tube has been inserted. Very little change has taken place as yet in the number of distended loops of small bowel. Conclusion: Cantor tube in jejunum. Mechanical small bowel obstruction (Fig. 2).

FIG. 2. Case 1. Intestinal decompression tube well down in the jejunum. Again note the characteristic pattern of small intestinal distention. No gas in the colon.

As a result of treatment with intubation, antibiotics and the restoration of a normal fluid and electrolyte balance, the patient improved daily. On the eighteenth hospital day, he developed a fluctuant mass in the pelvis which ruptured into the rectum, discharging a large amount of foul purulent material. His recovery from that day on was rapid. He was discharged as cured on June 3, 1949, his twenty-seventh hospital day.

The final diagnosis was: pelvic abscess with paralytic ileus.

Comment. This patient presented all the characteristic roentgenological criteria for a diagnosis of small intestinal obstruction. A roentgen diagnosis of mechanical small-intestinal obstruction was made and operation advised. On clinical grounds a diagnosis of paralytic ileus due to pelvic abscess appeared most likely. Despite the absence of gas in the colon, it was felt that an unusual gas pattern was present in this case, and that it constituted an exception to our usual roentgen diagnostic criterion. The final diagnosis of paralytic ileus was proved by the clinical course of the disease. Surgical intervention in this case might have been most serious.

Case II. A.A., a female, aged fifty-five, was admitted to the hospital on July 25, 1949, complaining of cramping pain in the abdomen and vomiting. The pains began insidiously in the mid-portion of the abdomen about ten days prior to admission. Vomiting followed shortly thereafter. She had vomited almost all of her food for the past week. She noticed that she had been more constipated in the last month and has had no bowel movement for several days before entering the hospital. She had never noticed any bloody or tarry stool although she had recently had some trouble with hemorrhoids which she attributed to straining incident to the episodes of vomiting.

Past history by systems was negative.

Physical examination showed no significant findings except for the abdomen which was distended and tympanitic but not tender. No fluid wave or masses were detected. Peristaltic sounds were diminished and heard only in the right lower quadrant of the abdomen. Pelvic examination showed no evidence of pathology. Rectal examination showed a grade II internal and external hemorrhoids. Digital examination showed the rectum to be filled with soft feces. After adequate preparation, a sigmoidoscope was passed 25 cm. No mucosal lesions were found. A preliminary film of the abdomen on July 26, 1949, showed considerable gaseous distention of the small intestine with no gas visible in the colon. Roentgen diagnosis: Marked low obstruction of the small bowel-ileum (Fig. 3).

Fig. 3. Case II. Note the marked small intestinal distention with absence of gas in colon. Carcinoma of the sigmoid found at operation.

On July 27, 1949, the patient's abdomen was opened. The entire small intestine was found to be dilated down to the ileocecal junction. The cecum contained only a small amount of gas while the ascending and transverse colon which was immediately visualized was contracted and empty. While the cecum was being palpated, the gas from the ileum passed in and caused it to dilate up to the hepatic flexure. This area was then mobilized in search of a tumor and during this manipulation the transverse colon filled with gas. Palpation was then continued down the descending colon which was still contracted and contained soft stool which increased in amount down to the mid-sigmoid. There a napkin-ring type of tumor was found to cause a high grade obstruction. The bowel below the tumor was collapsed and empty. By the time this exploration was completed, the entire colon down to the tumor in the mid-sigmoid was distended with gas. A transverse loop colostomy was done and the wound closed. No drain.

Diagnosis: carcinoma of the sigmoid colon.

On August 22, 1949, a resection of the

sigmoid colon and an end-to-end anastomosis was done. This was followed by a resection of the stoma of the colostomy and anastomosis on September 2, 1949.

Comment. This patient was proved to have an annular obstructing carcinoma of the pelvic colon. Roentgenologically she should have shown considerable distention of the colon with or without gas in the small intestine. Actually, however, she presented the roentgenological findings of a marked small intestinal distention with no gas in the colon so that a roentgen diagnosis of small intestinal obstruction was made. At operation, the colon was found to be collapsed and free of gas while the small intestine was dilated. It is difficult to explain this condition on a mechanical basis. The intestine was apparently in a sympatheticotonic state with persistent spasm of the ileocecal valve and a hypotonic and dilated small intestine. As soon as the ileocecal area was palpated in searching for the cause of the obstruction, the gas began to pass into the cecum and through the colon to the point of obstruction in the sigmoid. The absence of gas in the colon was probably due to the fact that she passed considerable gas following an enema which had been given before the preliminary film was taken. Such a sympatheticotonic state is not commonly associated with obstruction in the left colon.

SUMMARY

Intestinal gas patterns have been studied for many years. The radiologist can usually diagnose the presence of a small or large intestinal obstruction from this alone. A preliminary film often is sufficient to indicate to the radiologist that an obstructive lesion is present. It must be remembered, however, that this method is not without error. At times bizarre gaseous intestinal patterns appear that tax the diagnostic acumen of the radiologist to the utmost. In such cases, further study by barium enema or small intestinal study by means of barium suspension injected through an indwelling long intestinal tube would seem indicated before a definite diagnosis was made.

The 2 cases presented show the fallacy of diagnosing all cases presenting small intestinal distention without gas in the colon as mechanical small intestinal obstruction. Although in the majority of cases such findings would occur with a mechanical obstructing lesion of the small intestine, we must be alert to the possibility of the gas pattern being misleading. Grave consequences to the patient can only be avoided in such cases by the close collaboration between radiologist and surgeon with a proper appreciation and evaluation of the physical findings.

CONCLUSIONS

1. Two cases are presented to exemplify a type of case in which roentgenologically all the signs of small intestinal mechanical obstruction were shown, but in whom no small intestinal obstruction was found.
2. Small intestinal distention without gas in the colon can occur without small intestinal mechanical obstruction.
3. Occasionally small intestinal distention without gas in the colon will be found in paralytic ileus or as a result of an obstruction of the pelvic colon.
4. No enema should be given to any patient prior to a preliminary film of the abdomen if a proper evaluation of the gas pattern is to be made.

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BIOLOGICAL EVALUATION OF 20 MILLION VOLT ROENTGEN RAYS*

V. BAR EFFECT IN DROSOPHILA

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THERE are two main reasons for comparing responses to high and to low energy roentgen rays: in radiotherapy, the utilization of high energy rays will be facilitated if one knows how to transfer experience acquired with conventional radiations into the high energy field; in radiobiology, differential responses to different radiations are used to learn something about the reacting entities. For either case, it is desirable to have a broad experimental basis with several and diverse reactions. Any measurable response to radiation is usable for this purpose, and, at present, it is not possible to say which responses will be the most useful ones.

The "*bar-eyed*" character in *Drosophila melanogaster* is defined by a reduction in the number of eye facets. Several kinds of *bar*, distinguished by the degree of facet number reduction, occur as inherited anomalies. They are thought to be connected with duplication of a certain chromosomal segment. The effect of the genetic anomaly can be copied by a number of treatments which must be administered during a certain sensitive period of the larval stage. Treatments with graded stimuli such as temperature[1] or roentgen rays[3] produce a graded reduction of the facet count. Consequently, the eye of *Drosophila* can serve as a biological dosimeter, the readings of which, compared to those obtained with a thimble chamber, can be used to estimate the efficiency of any radiation tested:

$$\eta(t@s \| Y,u) \equiv D(Y,u,s) \div D(Y,u,t)$$

where η is the efficiency of the radiation tested, t, compared to the standard radiation, s, Y the biological response and u the dosage unit on which the comparison is based, and D the doses of standard and test radiation in units u, needed to elicit the response Y.[5]

Previous determinations of η (20 million volt @ 200 kv.) with various biological responses to a single irradiation have given values of about 0.75; the present test with the *bar* effect falls in line with previous results. *Drosophila* have been used once before in this program.[2] At that time, radiation was administered to adult males, and effects produced in the sperm were investigated by examination of the F2 generation. In the present study, the radiation was administered to larvae, and the effect noted in the irradiated animal after hatching from the pupa. Thus, the two *Drosophila* tests are quite different, and the results are independent of each other as far as biology is concerned.

MATERIAL AND METHODS

The flies used were an inbred strain of bar-eyed *Drosophila melanogaster*. They were kept at 28° C. during most of the period of development. This selection of strain and temperature gives a low facet number even before irradiation; thus the facet counts can be made with great accuracy and comparatively little labor.

The eggs were deposited on blobs of food on milk bottle caps. The larvae were irradiated on the bottle caps on which they had hatched. All irradiations were given during the "sensitive period" of the larval life,[3] though not necessarily during the interval of maximum sensitivity. The imagoes were preserved in 95 per cent alcohol soon after emergence. The facet counts were made by one of us (W.M.L.) who, at the time of counting, was not informed of the doses used. Only the right eyes of male flies were counted.

The source of the 20 million volt roentgen rays was the 22 mev. betatron of the University of Illinois at Urbana, Illinois. For each of the two tests made, presdwood was used to

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TABLE I

REDUCTION OF EYE FACET NUMBER IN DROSOPHILA AFTER IRRADIATION

Facet Count in Controls, and No. Control Eyes Counted	Quality of Radiation	Dosage Rate (r/min.)	Dose in r	No. Eyes Counted	Mean Reduction of Facet Number	Standard Deviation	Reduction of Facet Number per Roentgen		
							Single dose	Single technique	Single quality
46.4 in 110 controls	100 kv. no filter	1380	460	41	7.2	4.6	.0157	.0113	.0151
			920	46	10.7	3.7	.0116		
			1380	22	13.3	7.3	.0097		
		610	457	28	8.8	3.9	.0192	.0172	
			915	14	17.7	7.7	.0194		
			1373	24	20.1	10.6	.0146		
		160	480	3	8.2	5.5	.0171	.0171	
			960	6	17.7	6.1	.0185		
			1440	16	23.6	8.0	.0164		
	200 kv. no filter	106	422	34	5.5	4.5	.0130	.0154	.0135
			844	31	13.8	6.2	.0164		
			1266	17	19.9	4.2	.0157		
		400	400	37	6.0	7.6	.0150	.0104	
			800	10	3.9	7.9	.0049		
			1200	2	16.9	7.5	.0141		
	Betatron at 20 mev.	410	352	23	5.4	4.2	.0153	.0125	
			466	25	9.6	5.3	.0206		
			700	3	11.7	3.8	.0167		
			749	14	6.3	4.8	.0084		
			1215	15	13.1	6.9	.0108		
			1245	22	15.1	3.4	.0121		
46.5 in 54 controls	100 kv. no filter	1380	460	6	9.2	2.7	.0200	.0167	.0140
			690	9	6.4	5.3	.0093		
			1150	13	22.8	6.8	.0198		
		160	640	12	8.4	3.4	.0131	.0136	
			960	5	13.5	8.4	.0141		
		80	400	37	4.8	4.2	.0120	.0105	
			800	4	9.2	2.0	.0115		
			960	3	7.8	4.2	.0081		
	200 kv. no filter	106	633	9	3.2	3.5	.0051	.0091	.0127
			1055	5	12.7	9.5	.0121		
		57	456	16	5.8	4.2	.0127	.0146	
			912	18	11.3	5.1	.0124		
			1040	15	18.2	4.8	.0175		
	Betatron at 22 mev.	59	297	20	3.1	7.4	.0104	.0094	
			446	39	3.9	4.9	.0087		
			595	20	3.8	4.4	.0064		
			743	4	10.0	6.7	.0134		

build up electronic equilibrium at the position of the larvae. Thus the larvae were located in a region of fairly uniform intensity, behind the very steep dosage gradient of the transition region which occurs with high energy roentgen rays. The phantom and irradiation cell employed in the first test were those used earlier for the treatment of adult flies.[2] The average dosage rate was 410 r per minute. In the second test, a graphite filter, designed by L. S. Skaggs,[4] was used. This filter reduces the intensity of low energy rays in the beam and gives a uniform dose perpendicular to the beam. The bottle caps were stacked behind 3 cm. of pressdwood. The dosage rate was 59 r per minute.

Conventional machines for deep and superficial therapy were used to produce radiations with 200 and 100 kv. (peak) energy. No filters were used in either case. The dosage rates were varied by varying the distances.

The experiments were designed so as to be largely self-contained. All flies used were raised together, and assigned at random to the various techniques compared. The dosage rates used in both tests and with both standard radiations covered a considerable range which contained the rate used with the betatron. The post-irradiation treatment was the same for all flies.

RESULTS

A tabulation of techniques used, and of data obtained by counting 892 eyes, is given in Table 1.

The number of facets decreases linearly with dose (the tabulated value, i.e. the reduction of facet counts, increases accordingly). By inspection, most of the results could be fitted reasonably well by a straight line going through the normal facet count at zero dose. This impression was tested as follows: to each set of data obtained with one technique in one test, omitting the counts in unirradiated controls, a straight line was fitted by the least mean square method. The intercepts of the straight lines with the zero-dose-axis were calculated; the values so obtained are estimates of the normal facet number based on the results obtained in irradiated flies. The mean zero-intercepts were 44.1 ($\pm$0.9) in the first test, and 46.5 in the second, in good agreement with the values actually found in controls, which are 46.4 and 46.5.

The standard deviation within single sets of flies is, on the average, 5.3. A trend toward higher variance with increasing doses exists, but is negligible within the range tested. A "single set" comprises all flies raised and treated together (in a single test, with a single technique, with a single dose). Variations occurring within such a set are due to individual differences between flies, and to differences in dose and treatment; the latter differences were minimized by careful experimental technique.

The reduction of facet number per roentgen, or the ratio of the difference between counts in controls and irradiated flies, over the dose administered, should be a constant value if the effect is proportional to the dose. The table shows that this is the case; there are considerable fluctuations, but they are not systematic.

The second column of "reduction of facet number per roentgen" contains the estimates based on all flies treated in one test with one technique, though with different doses. One finds considerable fluctuation, but not in a systematic way. Of the techniques used, three were included in both tests, and, in each case, the two values obtained deviate from the mean in opposite directions. It appears that the dosage rate has no significant influence within the range tested. Therefore, the results obtained with different dosage rates were pooled to obtain estimates of the effects of each quality of radiation (last column of table). Pooling the results obtained with standard radiations in both tests, one obtains 0.0147 for 100 kv. (peak) and 0.0131 for 200 kv. (peak). Or, averaging for each test, one has 0.0153 for the first and 0.0135 for the second test. The differences between these values are rather small. Thus one may, in first approximation, pool the results obtained in both tests, with both standard radiations, all dosage rates and all doses, to obtain a weighted mean of 0.0141; this is the average facet reduction per roentgen,

using conventional types of radiation, under the conditions of our experiments.

The betatron tests gave reductions of facet numbers per "r" of 0.0125 and 0.0094, with a weighted average of 0.0119. The "r" is the reading obtained with a 25 r thimble of a Victoreen condenser r-meter, the thimble being inserted in the phantom at the same position as the irradiated objects, behind a layer of presdwood sufficiently thick to build up electronic equilibrium. The effect per "r" is less than the effects obtained with standard radiations; any dose in "r" of high energy rays is equivalent to a dose of $119/141 = 0.85$ r of standard radiation. Thus:

$$\eta(\text{20 million volt @ 100 or 200 kv.}\ Y = \text{reduction of eye facets in } \textit{Drosophila},\ u = \text{readings of r-meter}) = 0.85$$

There are a number of statistical tests which can be used to estimate the significance of the results given. We have used the following sequence: the means in each set were considered as estimates of the same "true" value, 0.0141. The deviations of the set means from this value were analyzed into two components, one due to the variance between individual flies (which is the variance within sets), and one due to factors operating on the whole set. The latter turned out to be far from negligible, and furnished an additional standard deviation of 0.0071 per set. The deviations of set means were again subjected to an analysis of variance, in order to separate factors operating on a single set from those operating on all sets belonging to a given technique; the latter would include errors in the calibration or in the output of a machine. This last component was found to be small (DEV. of 0.0012) and not significantly different from zero. The variances of the estimates of reduction of facet counts per r were estimated as the sum of the variances within and between sets, and adjusted to 1 r. Such variances were used to determine the weighted averages entered in Table 1.

The methods outlined in the paragraph above were used to estimate the standard deviations of the various differences in efficiency. The difference in effect between the two standard qualities of radiation was 0.0016, its standard deviation 0.0013; the difference between the two experiments was 0.018, with a standard deviation of 0.013; thus, both differences are suggestive but not certain. The difference in effect between all standard radiation and the betatron is 0.0022, with a standard deviation of 0.0014. Thus, provided the difference is of the order found, several additional tests would be required in order to obtain an estimate with good precision on this single test object. The result for η agrees well with the values obtained with other test objects.

DISCUSSION AND SUMMARY

The reduction of eye facet number in *Drosophila* after irradiation during the sensitive period was used to compare effects of high and low energy roentgen rays. It was found that η (20 million volt @ 200 kv.) $\sim$0.85, the amounts of both radiations being measured by a certain thimble chamber. The result falls in line with values previously obtained with responses to single doses: 0.79 for acute roentgen death in mice, 0.72 for graying of hair in mice, 0.75 for production of sex-linked lethals in *Drosophila*. None of these values was determined with very great precision; as far as statistics is concerned, they might be estimates of the same value. In order to obtain statistically significant differences, we would have had to experiment on a much larger scale. We did not feel it advisable to spend much effort in this direction at this time because the problem of measuring and defining doses of high energy roentgen rays is not a small one, and the comparison of doses in objects of different size and shape might easily be subject to systematic errors as large as the random errors which could be removed by a great effort. Thus, we consider tests

like those discussed in this paragraph as a part of a program of preliminary scanning of efficiencies over a range of radiobiological reactions, which will be made as wide as feasible. It must not be implied that there is such a thing as "the" biological efficiency of high energy roentgen rays. Efficiency is a figure which may (and sometimes does) vary with the response investigated, and even with the degree of response observed. Strong evidence in this direction has been presented,[5] and additional data are being collected.

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USE OF PRECISION CONES IN PERORAL IRRADIATION*

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ROENTGEN radiation delivered directly to an intraoral lesion constitutes an important irradiation technique. Judged by the meager contributions to the literature in the past twenty years, no considerable effort has been directed toward a refinement of peroral technique. The purpose of this communication is to present a method of peroral irradiation which permits the therapist to achieve precision in technique not possible by previously described methods.

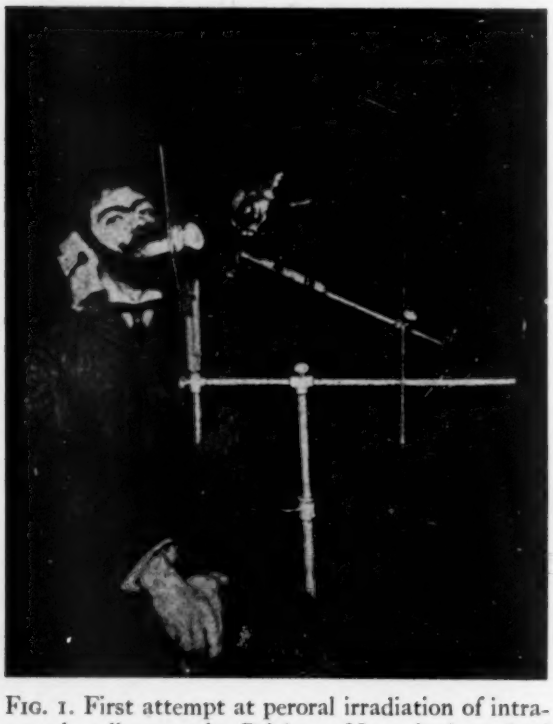

FIG. 1. First attempt at peroral irradiation of intraoral malignancy by Brickner. Note the large lead shield.

Peroral roentgen irradiation of intraoral malignancy was perhaps first attempted by Brickner[2] in 1903. In that year he treated a malignancy of the buccal mucosa, using a Ferguson vaginal speculum placed through an opening of appropriate size in a large sheet of protective lead (Fig. 1). The vaginal speculum was placed through the opening and into the mouth in such a manner as to expose the intended area to direct roentgen treatment. Because of technical difficulties involved, Brickner's lead was not followed. Intracavitary roentgen therapy was delayed until the development of shock-proof, ray-protected, flexible, compact tube heads.

Not until 1937, when Martin[5] described his technique for peroral irradiation, did direct irradiation of intraoral lesions receive much further consideration. Martin's contribution undoubtedly influenced the management of intraoral malignancy. Metal cylinders of brass tubing in various sizes (2.5, 3.0, 3.5 and 4 cm. in diameter) were attached to a master cone in such a way that the central ray of the roentgen tubes lay in the prolongation of the axis of the cylinder. Martin's technique, also used or rather closely followed by Merritt,[6] Dresser and Dumas,[3] and others has probably become fairly well established in many institutions, as a standard for peroral therapy.

Ebenius[4] in 1941 described a method of peroral irradiation whereby the treatment cylinder is stabilized against teeth and gums by dental compound impressions. This technique was developed to overcome the danger of motion on the part of the patient during treatment. The warm and pliant dental compound is attached to the end of a treatment cylinder. While the compound is still soft, the cylinder is placed in the desired position and when the patient bites, an impression is obtained of the teeth and jaws. In this way the cylinder becomes firmly fixed in its relation to the tumor. In our judgment this technique

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has limited applicability and, most important, does not permit protection of those parts of the oral cavity which do not require irradiation.

Baclesse[1] in 1947 described a 100 kv. short distance apparatus for endobuccal roentgen therapy. This apparatus consists of a Tubixol tube which can be rotated through 360° in the frontal and sagittal planes with a target angle of 32° and a special device for holding the various sized cones stationary. It is used at the Curie Foundation in Paris and provides a form of treatment intermediate between endobuccal contact roentgen therapy and deep roentgen therapy. This technique has limited adaptability as to location and thickness of lesions. Although the cone is probably held fairly stationary, there is no fixation between the cone and the tube head. Thus, in our opinion, the use of a modern tube head constitutes the only significant improvement upon Brickner's original contribution. Although fixing the cone rather than the entire apparatus facilitates patient set-up when vernier control is unavailable, inherent dangers in this method do exist. This method, as that of Ebenius, does not provide adequate protection to those parts of the oral cavity which do not require irradiation.

The foregoing constitute the only significant contributions dealing with peroral roentgen therapy found in available radiological literature.

In 1943, one of us (S.G.C.) first conceived the idea of fashioning individual treatment "cones" for each and every intraoral lesion to be treated, and since then we have administered over 4,000 treatments by this method. The "cones" are "tailor-made" for each patient. No two cones are alike. The fundamental technique is relatively simple, although innate manual skill is essential. First, one must have a set of master cones, oval and round, varying in diameter from 2 cm. to 6 cm. These master cones are 6 cm. in length and with attached special cone, when connected to the treatment perisocpe, give a target skin distance of over 36 cm., depending upon the length of the special cone. The master cones all have a rim or shoulder upon which the special lead cones rest (Fig. 2). The individualized "cones" are made of sheet lead 1/16 inch in thickness. A strip of lead about 6 cm. in length and about 1 cm. wider than the circumference of the cylinder is wrapped around the master cylinder which has been selected for the case (Fig. 3). After wrapping the lead about the cylinder, the distal end of the lead cone is fashioned with small tin shears so as to conform to the parts in the oral cavity with which contact will be made in delivering the treatment. The sharp edges resulting are then smoothed with the blunt side of a knife or some dull-edged instrument. A sharp blade, unless used very painstakingly, will raise a "fur" rather than smooth the edge. The final polishing and burnishing are done with a mechanical buffer or manually with chamois cloth,

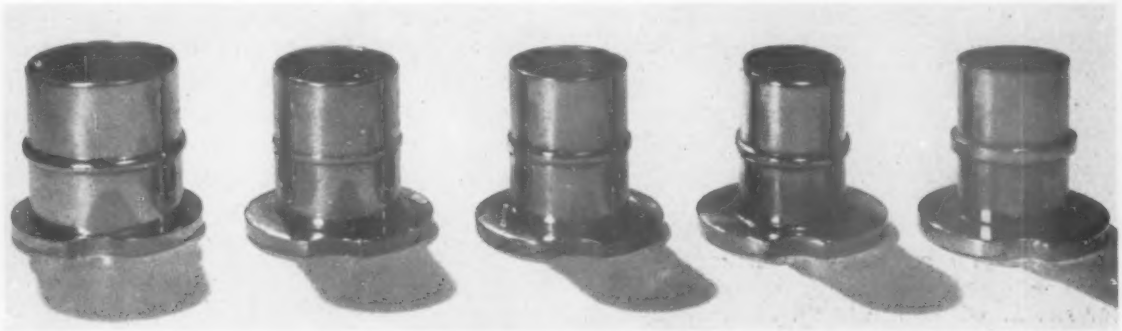

Fig. 2. Set of master cones.

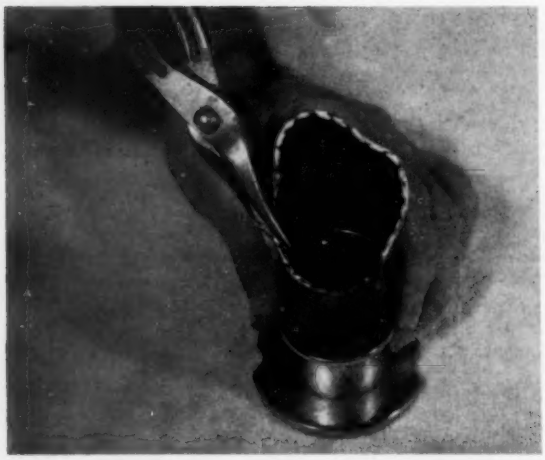

FIG. 3. Fashioning cone. The edge must be brought to a high polish.

canvas or other material. Several fittings are made with the patient *in the treatment position* and various modifications are made as they appear indicated. The treatment position of a given patient is variable, depending upon the location of the tumor. The treatment position of the cone is constant, i.e., vertical. Several sand bags may be required under the patient's occiput to flex the neck on the chest (for treatment of floor of mouth lesions). Sand bags may be placed under the patient's shoulder and buttocks to throw his head back in order that a palatal, superior sulcus, antral, nasal cavity or nasopharyngeal lesion may be treated—always remembering to position the lesion so that the roentgen-ray beam, projected vertically, strikes the surface of the lesion perpendicularly or as nearly so as possible.

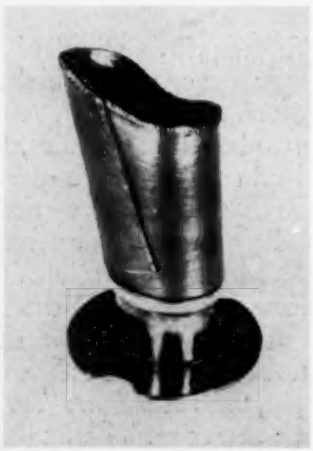

FIG. 4. Flaring effect. The overlap is corrected.

Often a flaring effect is desirable at the end of the cone (Fig. 4). It should be emphasized, however, that the useful flare effect is limited by the columnation of the roentgen-ray beam. Based on cones of average diameter and length, a flare effect which increases the diameter by 0.5 cm. is about maximum. This flare effect takes up the original excess width distally. The proximal excess is cut away and the two edges of the lead cone are properly fitted from the proximal end to the flared distal end. Although further flaring in one direction or another may be indicated for purposes of retraction, care must be exercised in appreciating the limitations of the useful roentgen-ray beam in such cases. The last step is to fasten the completed cone to the master cone by means of adhesive tape. As treatment progresses, it is often advisable to modify the shape of the cone so that it continues to fit properly. Occasionally, the open end of the cone is narrowed to concentrate higher doses to a resistant residue. After the completion of a treatment, the lead cone is easily removed from the master cone, cleaned and filed for use the following day. The master cone is sterilized for the next case in which it is required.

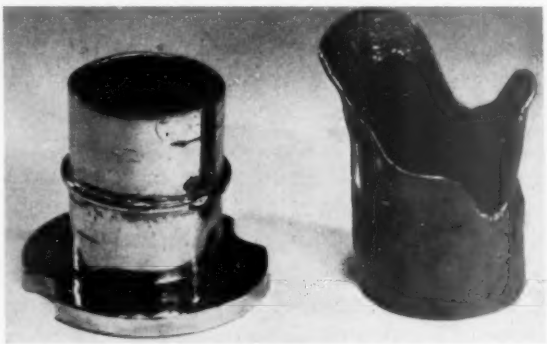

FIG. 5. A completed cone with its adapter.

When the cone is completed, the entire circumference of its distal end makes

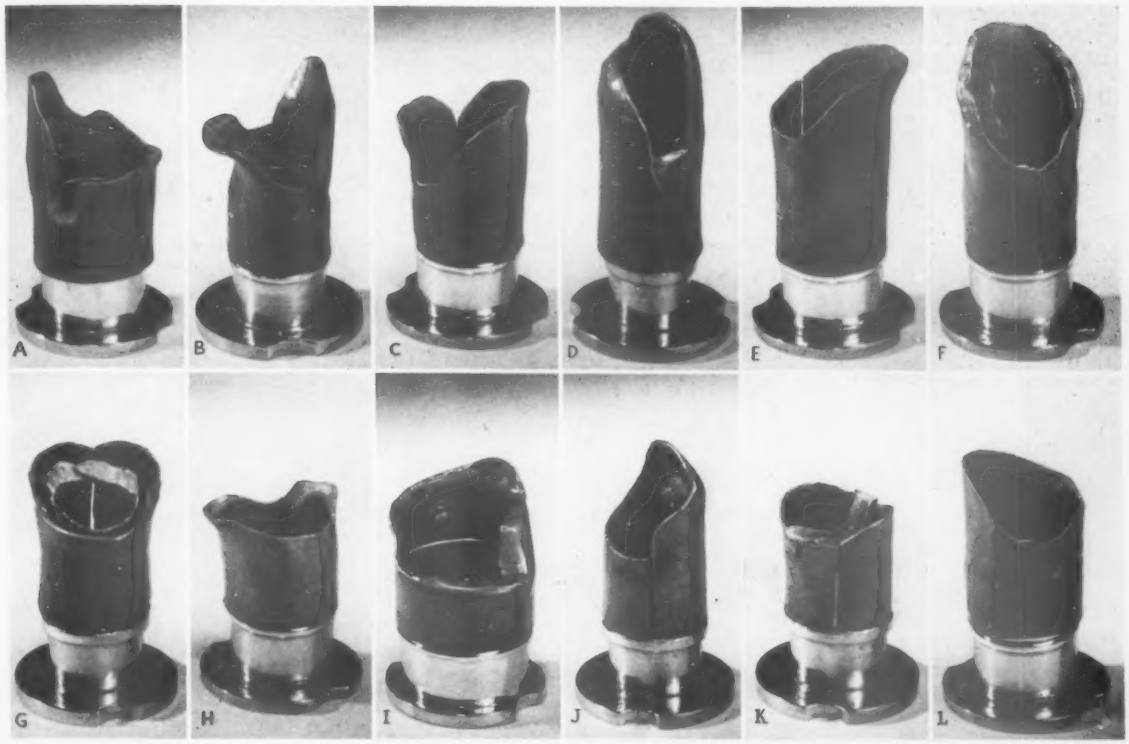

Fig. 6. Illustration of various shaped cones. (*A*) nasal cavity; (*B*) left floor of mouth; (*C*) right tonsillar fossa and soft palate; (*D*) gingival ridge (*E*) left submaxillary salivary gland; (*F*) hard palate; (*G*) anterior floor of mouth; (*H*) upper incisor alveolar ridge; (*I*) right maxillary antrum; (*J*) right side of tongue; (*K*) anterior floor of mouth; (*L*) left upper gingival buccal sulcus. The lead cones are attached to the master cone by means of adhesive tape.

reasonably close contact with the oral mucous membrane. The modified distal end of the cone assists in stabilization and affords protection wherever possible. Only simple equipment is needed, such as various sized ballpeen hammers, 6-inch tin shears, smooth-jawed pincers, a knife, and buffing material, Each lead cone is shaped for its special purpose—grooved or notched for an alveolar ridge, flanged and/or rolled to push a cheek or tongue aside, or shaped to protect all or a segment of a mandible.

By proper use of these "tailor-made" cones, it is often possible to avoid or greatly reduce undesirable pharyngeal irradiation in the peroral treatment of tonsillar and other more posteriorly placed lesions. In the treatment of lingual and floor of the mouth lesions, the alveolar ridge can be protected in an amazingly simple manner. In the preoperative treatment of lesions of the antrum or nasal cavity, the treatment can be so accurately delivered along the midline that healing (following resection of the palate as part

Fig. 7. Technique for introduction of cone and lowering periscope.

of the surgical management) is almost by primary intention; no trouble whatever is experienced with devitalized bone. In general, it may be said, irradiation is confined to known disease and an adequate margin of adjacent apparently normal tissue, while protection to surrounding vital structures is made possible.

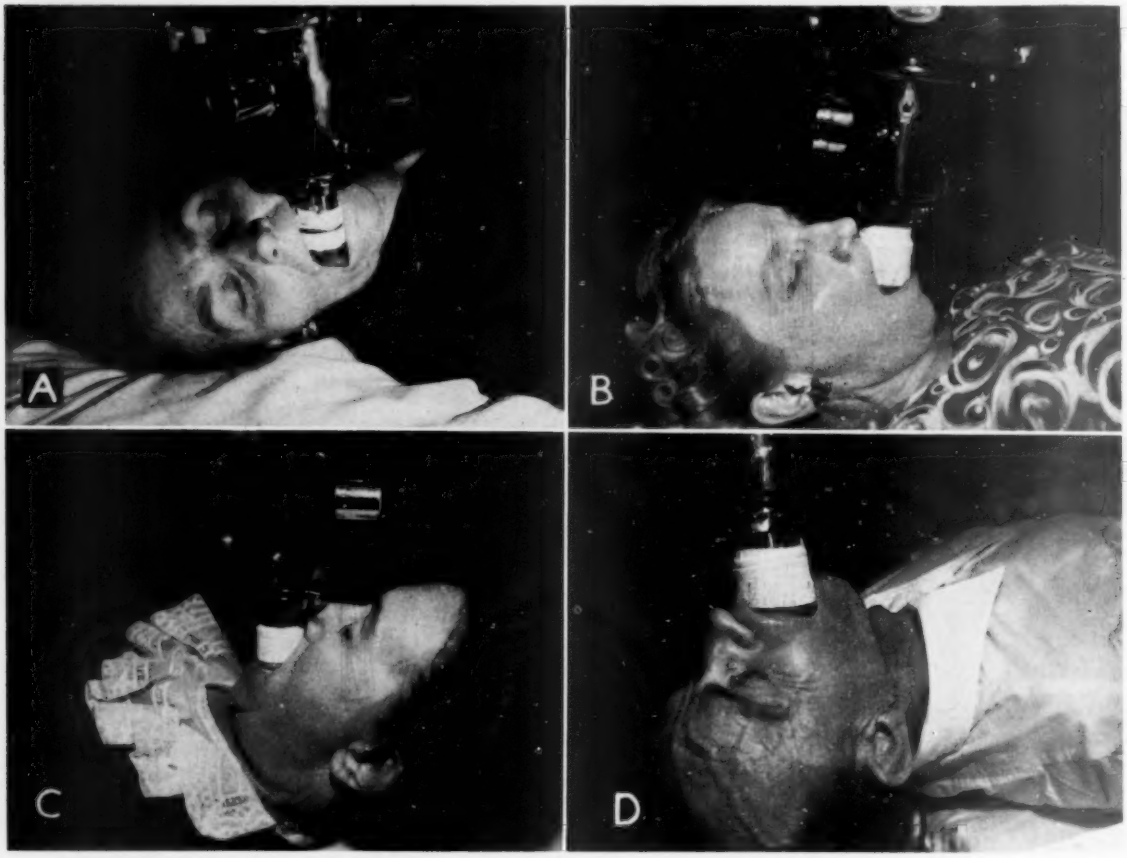

FIG. 8. Various treatment positions. (*A*) buccal surface lesions; (*B*) posterior floor of mouth; (*C*) anterior floor of mouth; (*D*) palate lesion.

Technique for the Introduction of the Cone. Various methods have been used in developing the present technique. It seems unnecessary to review the steps which have led to the technique used at present.

The patient is placed on the treatment table and the head, immobilized with sand bags, is placed in the same position used in the development of the cone (the treatment position) so that the rays will be directed towards the tumor area in a vertical manner. This eliminates gross adjustments and requires a minimum of vernier control. The cone is first placed in the patient's mouth and properly seated. The cone is then held in place while the periscope, which is fastened to the tube head, is lowered carefully and attached to the open end of the master cone. The position is then checked by the use of the periscope. On termination of the treatment the position is again checked.

CONCLUSION

It is not the purpose of this paper to defend peroral irradiation, although judging from growing sentiment and expressions in some quarters, a contribution with such a purpose would be timely. Intraoral carcinoma lends itself favorably to curative

irradiation efforts, not so much because of the radioresponsiveness of these lesions, but, rather, because of their accessibility. They present to the therapist the opportunity for direct treatment and direct observation of the effects of this treatment. In our experience the use of "tailor-made" lead cones individualized for each lesion permits more precise therapy of intraoral carcinoma. By irradiating the tumor and only an adequate margin of surrounding apparently normal tissue, volume dose can be more effectively controlled. Undesired irradiation of adjacent and neighboring tissue can to a large extent be eliminated.

By the use of the technique described, tumors of the tongue, buccal surface, tonsil, gingiva, hard and soft palate are in a sense converted into surface lesions and can thus be treated in a manner similar to skin tumors, where a beam of sharply screened or delimited roentgen rays strikes the tumor without first traversing an overlying layer of normal tissue. Because of the sharp and accurate delimitation, we are able to make possible full use of fractionation, protraction and protection, thereby greatly decreasing the incidence of morbidity. Radiation osteonecrosis has ceased to be a problem in our department. Finally, and most important, tumor dosage can be safely increased, with a correspondingly increased likelihood of tumor sterilization.

Photographs by Mr. A. V. Mucha, Temple University School of Dentistry and Mr. Robert L. Beyers, American Oncologic Hospital.

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EXPERIMENTAL NEUROGRAPHY*

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PRESENT diagnostic methods make it difficult and often impossible to locate a lesion of a peripheral nerve or nerve root, and, if located to determine whether it is amenable to surgical treatment or not. The development of a technique for the roentgen visualization of these structures by means of an innocuous contrast medium would advance the solution of this important problem.

Both experimental and clinical searches for such a medium have already been made. One of the first attempts to visualize peripheral nerves roentgenographically was made by Nikoloajev and Rubinstein[12] (1928). They obtained neurograms by injecting collargol, a colloidal silver compound, into the sciatic nerves of dogs.

Later Ogata[13,14] undertook a series of investigations. Among the substances tested were a variety of lead, mercury and iodinated organic compounds as well as lipiodol and lothian. With the exception of lipiodol they all proved to be rather toxic.

Sullivan and Mortensen[20] made neurograms of the sciatic nerves of rabbits. By injecting 0.05 cc. of a light brominized oil directly into the nerve fasciculus, they obtained satisfactory roentgen visualizations. Successful sciatic neurograms were also obtained in rabbits by Mulder,[10] who used 0.05 cc. of jodipin (Merck). These investigations,[10,20] however, did not include functional and histologic studies.

French and Strain[5] using pantopaque, reported and illustrated successful neurograms in rabbits and dogs. Although they did not record functional and histologic results they state that the "animals showed no ill effects from the injections."

Saito and his co-workers[17] applied the technique of neurography to man, using an emulsion of iodized oil and honey, which they called l'ombre. They made intraneural injections of 0.5 to 1.5 cc. of l'ombre into seven nerves (peroneal, sciatic, tibial, radial, ulnar and median nerves). Roentgen visualization of the nerves was obtained for distances varying from 7 to 12 cm. upwards from the injection site. Absorption of the oil, they reported, was almost complete in four to seven weeks. Reproductions of their neurograms showed rather faint delineation of the nerves. In 3 additional patients they made epidural sacral injections of 5–20 cc. of l'ombre and visualized several centimeters of the sacral nerve roots and the plexus. Detailed tests of function in these patients were not recorded, but the investigators state that "no unfavorable symptoms followed during and after the injection, such as pain during the injections, neuralgia or paralysis." Better neurograms were later obtained by Saito,[18] using 0.5 cc. of thorotrast. In this way he visualized the ulnar nerve for distances varying from 7 to 39 cm. upwards from the site of injection.

Since then many investigators[2,3,7,8,9,11,15,19] have used thorotrast as a neurographic agent in man. Studies of the absorption of this contrast medium and of its possible toxic effects over an extended period were not part of these investigations.

The dangers inherent in this radioactive compound have been repeatedly stressed. An editorial[23] in the *Journal of the American Medical Association* states: "It is important to remember, however, that degradation products of thorium emit alpha rays more penetrating than those of the radium series and that this ray is about 10,000 times as toxic to tissues as the gamma ray which is used therapeutically. . . . Experience with radioactive intoxications

* From the Neurosurgical Service of The Jewish Hospital of Brooklyn and the Department of Neurosurgery, New York Medical College.

indicates that as much as ten or fifteen years (and perhaps longer) may elapse between ingestion of the active material and the onset of gross tissue changes. The fact that patients who have been injected with thorium dioxide have shown no detectable toxic effects from the substance over a period of a few years is not satisfactory ground for complacency as to ultimate results. Recent reports, on the contrary, emphasize the proclivity of this substance to produce necrosis and malignant change in tissues with which it remains in contact." For these reasons the use of thorium compounds for clinical neurography is contraindicated.

Rottgen[16] used perabrodil for the visualization of peripheral nerves in clinical practice. The photographic reproductions of his neurograms, however, are unsatisfactory.

Asenjo and his co-workers[1] used iodized poppyseed oil (lipiodol) clinically in an attempt to visualize the site of peripheral nerve lesions. Judged by their two figures, the results were not very successful.

Clinical neurography is coming into increasing use. Experimental studies have largely overlooked the functional and histopathologic effects of the agents employed for delineating nerves. It therefore seemed desirable to continue studies of various contrast media with particular emphasis on functional and histopathologic effects as well as on the quality of the roentgenographic visualization. This is the purpose of the present study.

METHODS

Eleven radiopaque compounds, including pantopaque and several of its emulsions, were investigated. They were injected under direct vision into the sciatic nerves of rabbits. Ethyl iodophenyl-valerate and iodobenzene were not used, because French and Strain[5] have demonstrated their toxicity when injected into the sciatic nerves of rabbits and dogs.

During open-drip ether or intravenous nembutal anesthesia, the sciatic nerve was exposed through a mid-thigh incision separating the vastus lateralis from the biceps femoris muscles. The technique was aseptic throughout. The nerve was carefully cleared of its enveloping fascia. It was then elevated by means of a blunt nerve hook, and the injection material was introduced under slight digital pressure.

A No. 27 gauge needle, the tip of which had been bent to an angle of approximately 120°, was attached to a 1 cc. syringe. The needle, its bevel uppermost, was introduced through the superficial connective tissue sheath (chiefly epineurial, although doubtless in some instances perineurial also) and along the nerve for a distance of about 1 cm. At the first sign of extravasation of the fluid from the nerve, seepage back from the entry point of the needle, or marked local distention of the nerve, the needle was withdrawn. Reinsertion and withdrawal continued until the needle found a channel that permitted a free flow of the medium along the nerve.

Over-distention of the epineurial sheath with a great quantity of the injected compound is of no value in obtaining a satisfactory neurogram. The introduction of the material directly into the parenchyma of the nerve is also undesirable, because greater damage to the nerve fibers results and the chances for successful neurography are not improved.

Injections were made at a point midway between the head of the great trochanter and the popliteal fossa. Usually the needle was directed centralwards. At the site of the great trochanter of the femur, the nerve is sufficiently taut to permit injection without elevating it on a nerve hook. Injections made in this way, without handling the nerve, minimize the risk of trauma.

The opposite hind limb of the animal, serving as a control, was injected with a quantity of saline solution or of air equal to that of the contrast medium. In some cases, the control nerve was handled as for injection but without any actual injection.

If the injection impaired function, daily functional tests were made, usually until return of function occurred. Thereafter these tests were made at weekly intervals. Motor power was studied by observing the animals' gait and also by means of the spread reflex, elicited by suddenly lowering the suspended animal. The reflex disappears after complete anatomic or physiologic interruption of the sciatic nerve. The lessening of the amount of spreading of

the toes, which may be graded in several degrees, indicates roughly the degree of impairment of nerve function. Pain sensation was studied by pricking the foot with a pin.

Roentgen studies were carried out immediately after and, in some cases, during the nerve injections. They were repeated at intervals varying from several minutes to several weeks. In most cases the rabbits were not sacrificed until they showed full functional recovery as well as absorption of the injected material.

At necropsy* the sciatic nerves were removed and segments were fixed in 10 per cent solutions of formaldehyde. Hematoxylin and eosin as well as myelin-sheath stains (Spielmeyer) and axis cylinder impregnations (Gros-Bielschowski) were obtained. Scharlach R preparations for fat were also made.

RESULTS

Water Soluble Compounds

I. *Argyrol, 50 per cent* (mild protein silver. A. C. Barnes Co., Philadelphia).

Two-tenths of 1 cc. of argyrol was injected into the sciatic nerve of 1 rabbit. During the injection the animal kicked violently. Postoperatively there was a complete paralysis of the injected limb. The nerve was visualized poorly by roentgen ray. Post-mortem study thirty-nine days after operation showed dense adhesions between muscle and nerve. Microscopically there was evidence of complete destruction of the nerve fibers.

II. *Protargol, 15 per cent* (silver albumose. Winthrop Chemical Co., New York).

In the animal used for the above experiment, 0.2 cc. of protargol was injected subepineurially into the contralateral sciatic nerve. Afterwards the leg was fairly weak. The roentgen visualization, while somewhat better than that obtained with argyrol, was still poor. Necropsy examination disclosed dense adhesions of the nerve to the surrounding tissues. Microscopic examination revealed extensive destructive changes of the nerve.

III. *Hippuran, 50 per cent* (sodium ortho-iodo-hippurate).

The injection of 0.2 cc. of hippuran into the sciatic nerve of 1 rabbit resulted in violent kicking movements and muscular fibrillations. Total paralysis of the limb followed. Edema of the limb persisted until necropsy, forty-two days later, when there was edema of the nerve trunk and marked engorgement of the epineurial veins. The roentgenographic visualization of the nerve was poor. The control, nerve, injected with air, also roentgenographed very poorly. Microscopically there was extensive destruction of nerve fibers, which were almost completely replaced by fat-filled phagocytes.

IV. *Skiodan* (sodium iodomethan sulfonate, iodine content 40 per cent).

100 per cent solution. The injection of 0.2 cc. of skiodan was accompanied by vigorous kicking movements of the injected limb. There followed a complete paralysis of the limb, which persisted until death fifty-nine days later. Necropsy revealed considerable epineurial hemorrhage along the sciatic nerve and a number of fine adhesions.

The roentgen visualization of the nerve was very satisfactory. Microscopic examination revealed virtually complete destruction of the nerve. The epineurium was infiltrated with moderate numbers of monocytes and lymphocytes. The control nerve, injected with air, showed slight monocytic infiltration at the injection site.

75 per cent solution. Two-tenths of 1 cc. of skiodan injected into one sciatic nerve of each of 2 rabbits resulted in a loss of motor and sensory function in the injected limbs, which had returned to normal in each case within seven days. Roentgen visualization of the nerves was fair. The animals were sacrificed after seven and ten days, respectively. Grossly, one nerve appeared normal, but in the other there were marked epineurial hemorrhages at the site of injection. Moderate degeneration of nerve fibers took place.

50 per cent solution. In 1 rabbit, 0.2 cc. of a 50 per cent solution of skiodan produced no functional abnormalities, but provided inadequate roentgen visualization. The animal was sacrificed after twenty days. Microscopic examination showed well marked degeneration of nerve fibers.

V. *Thorotrast.*

25 per cent concentration. One rabbit received an intraneural injection of 0.2 cc. of 25 per cent thorotrast. There was no functional abnormality, but at autopsy, twenty-five days after operation, there were thickening of the nerve

* Unless otherwise stated in the text, the functional as well as the gross and microscopic results in the control nerves were normal.

and adhesions to the surrounding tissue. The roentgen visualization was excellent in this (Fig. 1) and in 2 other rabbits injected with a mixture of thorotrast and india ink. The material migrated along the nerve distally to the knee and proximally to the lumbosacral nerve roots and the subarachnoid space.

Microscopic examination showed large collections of phagocytic cells laden with large refractile granules of thorotrast. These cells were present in the nerve sheaths as well as within the parenchyma of the nerve. They occurred intraneurially in long rows between nerve fibers. There was a striking lack of other cellular change, except some fibroblastic proliferation in the epineurium. Axis cylinders or myelin sheaths next to the thorotrast-containing cells showed no abnormality.

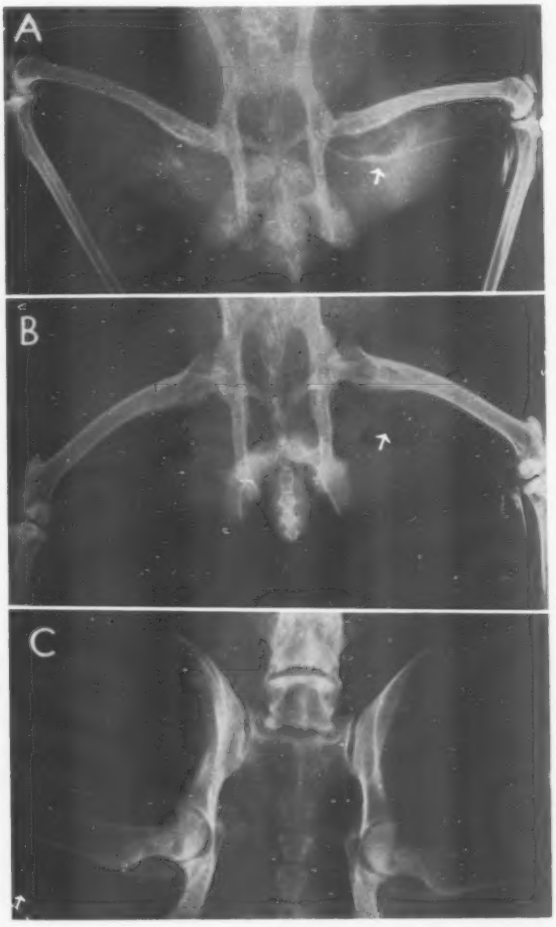

FIG. 1. Neurograms after the injection into the sciatic nerves of rabbits of (*A*) 0.2 cc. of thorotrast (25 per cent) and (*B*) 0.2 cc. of lipiodol. The injections were intraepineurial. (*C*) Neurogram showing the sciatic nerves and nerve roots of a large dog (the only dog used in this series of experiments) after the subepineurial injection of 0.2 cc. of pantopaque into each sciatic nerve. The contralateral neurogram was less successful. This animal showed no functional abnormality. The arrows indicate the site of the injections.

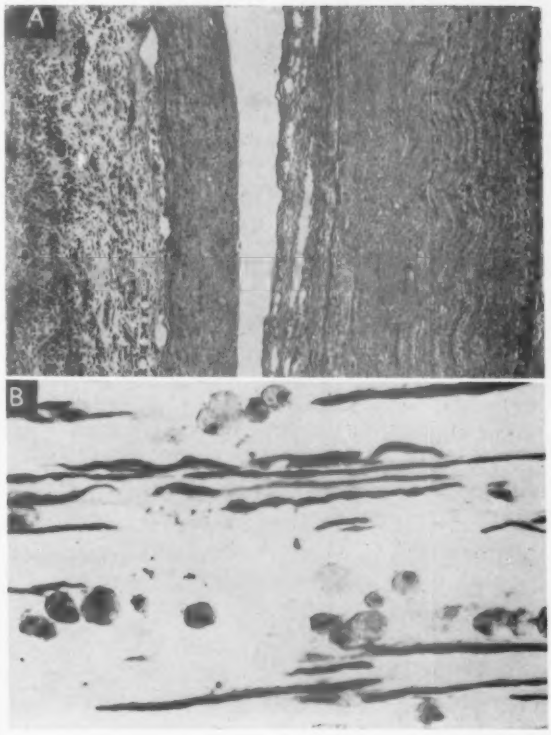

FIG. 2. Photomicrographs showing the microscopic appearance of sciatic nerves in which intraepineurial injections of 0.2 cc. of 12.5 per cent thorotrast were made. (*A*) shows considerable cellular infiltration and fibroblastic proliferation of the epineurium, in which there are many thorotrast granules contained in phagocytic cells. Hematoxylin and eosin stain, ×70. (*B*) shows thorotrast-laden phagocytes adjacent to nerve fibers. Gros-Bielschowski impregnation for axis cylinders, ×650.

12.5 per cent concentration. Injecting 0.2 cc. of thorotrast of this concentration into and around the nerve of 1 rabbit resulted in marked adhesions seen at necropsy twenty-five days later. The nerve was well visualized roentgenographically. No absorption of the thorotrast was noticeable on serial roentgenograms taken during the period of survival. Microscopic examination showed massive collections of granule-laden phagocytes both within and around the nerve trunk (Fig. 2). They were

most numerous in the perivascular spaces. Some of the axis cylinders were vacuolated and fragmented. Most of the nerve fibers, however, appeared to be intact.

6.25 per cent concentration. In 2 rabbits, injecting 0.2 cc. of thorotrast produced no functional abnormality during the survival periods of seven and fifteen days. Roentgen visualization of the nerves was satisfactory. No absorption of the material was observed throughout the survival periods. Postmortem examination of the seven day animal revealed marked thickening of the nerve trunk, with numerous petechial hemorrhages in the epineurium and many fine adhesions to the surrounding tissues. In the fifteen day animal, also, adhesions between nerve and surrounding tissues were seen. No abnormality of nerve fibers was seen, but the epineurium showed the same changes as the other nerves which received thorotrast.

VI. *Sodium Iodide.*

100 per cent solution. The injection of 0.2 cc. of sodium iodide into 2 rabbits was followed by complete motor paralysis and sensory loss of the injected limbs, which had not recovered when the animals were sacrificed forty-one and forty-three days later. Roentgen visualization of the nerves was good. Twenty minutes later the nerves were faintly delineated, and resorption was complete within two hours. Postmortem examinations showed dense scar tissue formation around the nerve trunk.

50 per cent solution. Two-tenths of 1 cc. of a 50 per cent solution of sodium iodide injected into 1 rabbit resulted in complete paralysis of the limb, which lasted for the twenty day survival period. This solution gave good roentgenographic contrast and was completely resorbed within fifteen minutes of its injection. Necropsy disclosed numerous fine adhesions around the nerve trunk.

25 per cent solution. Two-tenths of 1 cc. of this solution of sodium iodide was injected into the sciatic nerve of 1 rabbit. The animal showed partial motor and sensory loss and the formation of a trophic ulcer over the heel of the injected leg. Roentgen visualization was poor; and the material was absorbed within twenty-five minutes. Twenty days later the animal appeared functionally normal and was sacrificed. There were numerous adhesions about the nerve trunk, some rather dense. Many small petechial hemorrhages were present at the injection site.

20 per cent solution. Two rabbits were injected with 0.2 cc. of this solution of sodium iodide. On recovery from the anesthesia, they both showed a flaccid paralysis of the foot, which had lost the spread reflex and sensitivity to pin prick. Roentgen visualization was adequate immediately after injection, but ten minutes later none of the solution could be seen in the nerve. Slight recovery of the foot drop and spread reflex was apparent in 1 animal on the second postoperative day, when it was sacrificed. In the other animal, the palsy remained until the time of sacrifice on the eighth postoperative day. At autopsy the nerves appeared quite normal.

Histopathological examination of the nerves revealed little inflammation, but the fibers had degenerated badly as was the case in all of the nerves injected with sodium iodide.

15 per cent and 10 per cent solutions. Intraepineurial injections of these solutions into 2 rabbits provided insufficient roentgenographic contrast.

VII. *Neo-Iopax* (disodium N-methyl-3,5-diiodo-chelidamate; iodine content 51.5 per cent. Schering Corp., N.J.).

50 per cent solution. Injection of 0.2 cc. of neo-iopax into both sciatic nerves of 1 rabbit resulted in complete paralysis of the hind limbs, which had not recovered by the time the animal was sacrificed six days after operation. The roentgenographic contrast was good. The sciatic nerve between the knee and the head of the great trochanter was well outlined. Absorption was complete within one hour. Postmortem examination revealed extensive adhesions along the nerve trunk. Marked destructive changes of axis cylinders and myelin sheaths were evident on microscopic study. In a second rabbit, killed thirty minutes after a similar injection, marked epineurial hemorrhages occurred along the nerve. There were also hemorrhages within the nerve and marked disruption of nerve fibers.

25 per cent solution. Two-tenths of 1 cc. of this solution of neo-iopax was injected into one sciatic nerve of each of 3 rabbits. Two lost the spread reflex and showed foot drop for twenty-five and twenty-eight days, respectively. The spread reflex of the third diminished, and it lost the foot drop after six days. Roentgen visualization of the nerves was rather indefinite, and the solution was absorbed within forty-five minutes. The animals were killed after periods

of twenty-five, seventy-eight and eighty days, respectively. At autopsy extensive dense adhesions were observed around all of the injected nerves. There was marked degeneration of nerve fibers, which were largely replaced by fat-filled phagocytes.

VIII. *Diodrast* (diethanolamine salt of 3,4-diiodo-4-pyridone-N-acetic acid, iodine content 49.8 per cent. Winthrop Chemical Co. Inc., New York).

70 per cent solution. Each of 4 rabbits was injected with 0.2 cc. of diodrast. Functional observations were recorded for only 1 animal, in which foot drop and anesthesia of the injected limb were noted the day after injection. Roentgenographic contrast was fairly good. The major portion of the dye was absorbed within one hour, complete absorption requiring approximately seventy-five minutes. At autopsy, two to fifteen days after operation, there were fine adhesions along the nerve trunks and superficial hemorrhages in the epineurium. Microscopic examination of the injected nerves disclosed marked degeneration of the axis cylinders, consisting of fragmentation, thickening and vacuolization of the axones (Fig. 3*A*). Myelin sheaths were also vacuolated. There were subepineurial hemorrhages.

35 per cent solution. Two-tenths of 1 cc. of this solution of diodrast was injected into the sciatic nerves of 5 rabbits, and in 1 additional case it was infiltrated around the nerve. One animal suffered persistent foot drop. In another a trophic ulcer of the heel was still evident fifty days after injection. Functional observations were not made in the other rabbits. Roentgenographic visualization was adequate, and absorption was complete in one hour. Upon gross examination of these injected nerves two to 101 days after operation, they appeared normal. Microscopic study of the nerve of the rabbit in which the medium had been infiltrated around the nerve forty-one days previously disclosed no evidence of degeneration. Nine days after an intraepineurial injection, however, 1 rabbit showed considerable distortion of the nerve structure by hemorrhage, and the axis cylinders were thickened, vacuolated and fragmented (Fig. 4). Only occasional nerve fibers remained intact. The nerve of 1 animal, examined 101 days postoperatively, showed large accumulations of fat-filled phagocytes and signs of nerve regeneration. Two animals, which survived twenty-five and forty days, respectively, showed no histopathologic abnormality of the nerves.

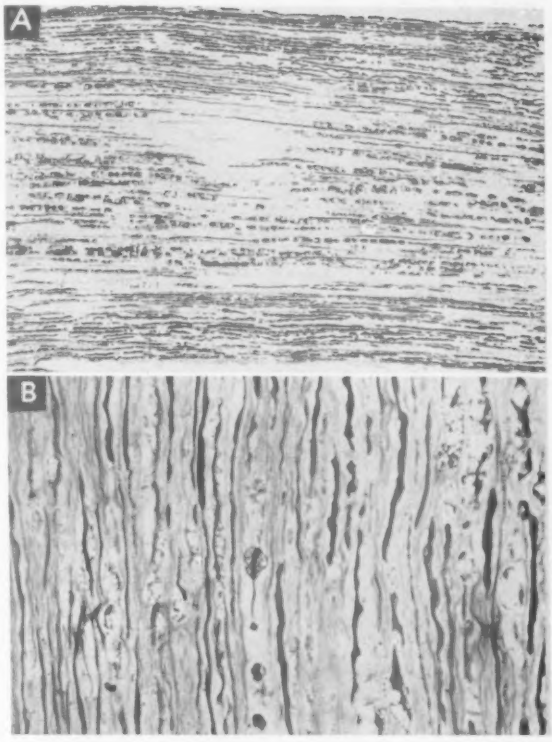

Fig. 3. Photomicrographs showing severe degeneration of axis cylinders produced by intraepineurial sciatic injections with certain contrast media. (*A*) shows marked degeneration, particularly in the central portion of the fasciculus. Diodrast (0.2 cc. of a 70 per cent solution) was used. The rabbit was sacrificed two days after injection. (*B*) shows axis-cylinder degeneration caused by the injection of 0.1 cc. of pantopaque emulsified chemically with a mixture of the Tweens.

Gros-Bielschowski impregnations for axis cylinders (*A*) ×95; (*B*) ×425.

17.5 per cent solution. Eight rabbits received intraepineurial injections with 0.2 cc. of this solution. Two showed a slight weakness of the injected limb immediately after operation. One of the 2 was entirely normal by the next day, and the other within one week. No other animals showed any functional abnormality. They were sacrificed three, five, nine, thirty-two, thirty-four, thirty-seven, forty-seven and seventy-three days after injection.

Adequate roentgenographic contrast was obtained provided that the films were taken immediately after the injection. Within fifteen minutes after injection, however, the opacity of

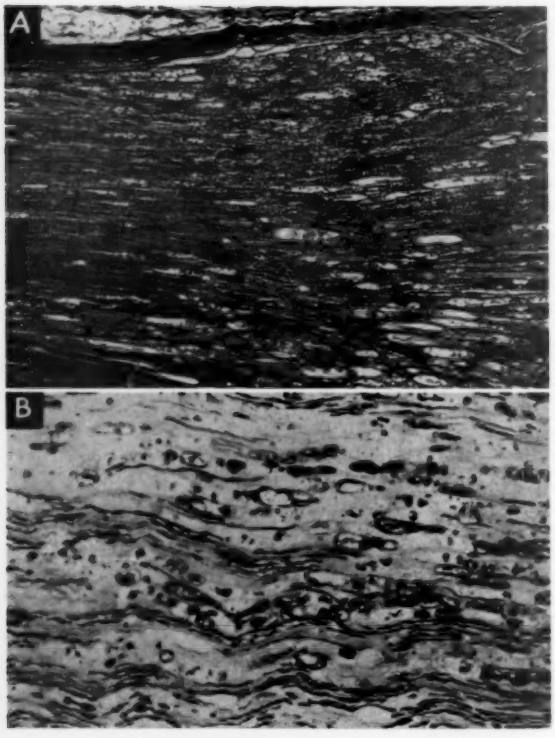

FIG. 4. Photomicrographs showing the sciatic nerve of a rabbit nine days after the intraepineurial injection of 0.2 cc. of diodrast (35 per cent concentration). The degenerative nerve fiber changes are especially clear in (*B*). (*A*) hematoxylin and eosin stain, ×95. (*B*) Gros-Bielschowski impregnation for nerve fibers, ×150.

the nerve significantly decreased, and the nerve was no longer visible after one hour.

Microscopic studies showed degeneration of nerve fibers, and, in some of the cases, numerous fat-filled phagocytes. These were rather marked in most of the nerves. Regeneration of axis cylinders was apparent in the older animals.

IX. *6-Iodo-D-Galactose* (iodine content approximately 49.8 per cent*).

Experience proved that this compound deteriorated rapidly on exposure to high temperatures or light, when it released free iodine, which rendered it extremely harmful to nerve tissue.

Intraepineurial injections of the sciatic nerves of 9 rabbits and 2 cats were made with 0.2 cc. quantities of fresh solutions of 6-iodo-d-galactose in concentrations of 40 per cent, 20 per cent and 13.3 per cent. The animals were allowed to survive for intervals varying from seven to fifteen days. Fairly good neurograms were obtained. Absorption of the medium occurred within approximately twenty minutes. Severe degeneration of nerve fibers, however, was a constant feature. The functional results were variable. With stored samples which had been allowed to deteriorate so that positive tests for free iodine were obtained, more severe functional and histologic reactions followed.

X. *Visco-Rayopake* (2,4-dioxo-3-iodo-6-methyl-tetrahydropyridine-1-acetic acid diethanolamine salt prepared in 71 per cent and 17.5 per cent aqueous solutions containing 10 mg. per cent zephiran chloride, specially prepared by Hoffman-LaRoche, Inc., Nutley, N. J.).

35 per cent solution. Each of 2 rabbits was injected with 0.2 cc. of visco-rayopake. Complete paralysis of the injected limbs followed. Good neurograms were obtained. Absorption of the compound occurred within twenty-five minutes. The animals were sacrificed after eleven and twenty days, respectively. In the first case, the spread reflex had reappeared and autopsy revealed an apparently normal nerve. Microscopic examination, however, showed rather marked degeneration of nerve fibers. In the second animal there was no evidence of functional recovery, and trophic ulcers were present on the heel. There was slight thickening of the connective tissue around the nerve trunk at the site of injection. Nerve fibers had degenerated rather markedly.

23.5 per cent solution. Three rabbits were injected with 0.2 cc. quantities of this solution. In 1 animal a slight muscular weakness followed. Recovery occurred by the time the animal was killed twelve days later. There was no evidence of impaired function in the other 2 animals, which were sacrificed two and five days after injection. Roentgen visualization of the nerve throughout the thigh was very good, and the dye was absorbed within about ten minutes of its injection. Necropsy on the first of these 2 revealed edema of the nerve trunk and prominent hemorrhagic extravasations in the epineurium. The nerves of the other 2 animals appeared grossly normal. Microscopic examination of the nerve removed two days after injection showed that most of the axis cylinders were thickened, fragmented and vacuolated. At five days, the degenerative changes involved the major portion of the nerve fibers, while at twelve days the axis cylinders of the

* The material was provided by J. D. Searle & Co., Spokane, Illinois.

entire trunk were in a severe degenerative state.

17.5 per cent solution. One of the sciatic nerves of each of 9 rabbits was injected with 0.2 cc. of this solution. No functional impairment followed. The neurograms were only fairly good. Absorption was very rapid, so that films taken ten minutes after injection revealed no trace of the material. The animals were sacrificed two, three, five, six, seven, nine, thirty-two and forty-seven days after operation. Slight and inconstant degeneration of nerve fibers occurred.

Water Insoluble Compounds

I. *Iodochlorol* (iodized-chlorinated oil. G. D. Searle & Co., Chicago).

Epineurial injection of this substance was not possible, since it was too viscous to pass through the lumen of a No. 27 or even a No. 23 gauge needle.

II. *Lipiodol* (lipiodol Lafay, iodine-addition product of poppy seed oil, iodine content 40 per cent. E. Fougera & Company, Inc., New York).

Lipiodol varying in amounts from 0.1 to 0.5 cc. was injected into one sciatic nerve of each of 5 rabbits. The injection was rather difficult because of the viscosity of this medium. Excellent visualization of the nerve occurred in 3 cases. The flow of the medium along the nerve was considerable in 1 rabbit (Fig. 1*B*). In this animal repeat films taken one month later showed that most of the lipiodol had left the nerve and migrated distally into the popliteal space.

Complete loss of spread reflex occurred in the limb which received 0.5 cc. lipiodol. Moderate impairment of this reflex occurred in 2 cases, the other 2 animals showing no functional abnormality.

Moderate infiltration of the nerve sheath and superficial nerve fibers with monocytes and lymphocytes occurred in the animal that received the largest injection. There was less cellular reaction in 2 rabbits that received 0.2 cc. lipiodol. The inflammatory changes were minimal in the 2 rabbits in which 0.1 cc. and 0.2 cc. quantities were used.

III. *Di-Iodo-Tyrosine, 25 Per Cent Suspension.*

Successful injection of this material proved impossible, since a suspension fine enough to pass through the lumen of a No. 27 gauge needle could not be produced in oil, water or rabbit serum.

IV. *Pantopaque* (mixture of ethyl esters of isometric iodophenylundecylic acids, iodine content 30.5 per cent. Picker X-Ray Corp., New York).

Six rabbits received intraepineurial injections of 0.2 cc. of pantopaque. In 4 of these animals there was no detectable functional abnormality throughout the respective survival periods of seven, sixty-seven, 177 and 203 days after injection. In 2 rabbits the spread reflex diminished moderately, then returned to normal at the end of six and 120 days, respectively. The first of these 2 animals survived seventy-five days, the other 402 days. In 1 of them the spread reflex diminished no more than on the control side. No paresis of the limbs was detectable in either animal.

Very good roentgen visualization of the sciatic nerves in the thighs occurred (Fig. 1*C*).

At necropsy there were fine adhesions at the injection site, but no more than on the control side. Microscopic studies showed either completely normal nerves (Fig. 5) or, in 2 instances, slight degeneration of a few nerve fibers, but no greater than on the control side. In one nerve moderate lymphocytic and monocytic infiltration of the epineurium occurred.

One rabbit received an injection of 0.1 cc. pantopaque. Fairly good roentgen visualization followed. There was no functional abnormality of the injected limb throughout the four day survival period. The nerve was normal histologically.

Another rabbit received 0.5 cc. pantopaque. Complete foot drop persisted for four days when the animal was sacrificed. Microscopically the nerve showed moderate lymphocytic and monocytic infiltration of the nerve sheaths and superficial nerve fibers.

Four rabbits received injections of 0.8 cc. of pantopaque. Two of these animals were completely normal throughout their fifty and 300 day survival periods. The 2 other animals showed foot drop of the injected limbs, which recovered at seventeen and thirty days. These animals were sacrificed at 130 and 240 days, respectively, after injection.

At necropsy adhesions at the injection site were greater than in the rabbits which received 0.2 cc. of pantopaque.

Microscopic examination showed slight rarefaction of the nerve in a few areas. The paralysis of the injected limb in the 2 rabbits was doubtless caused by nerve fiber changes. These

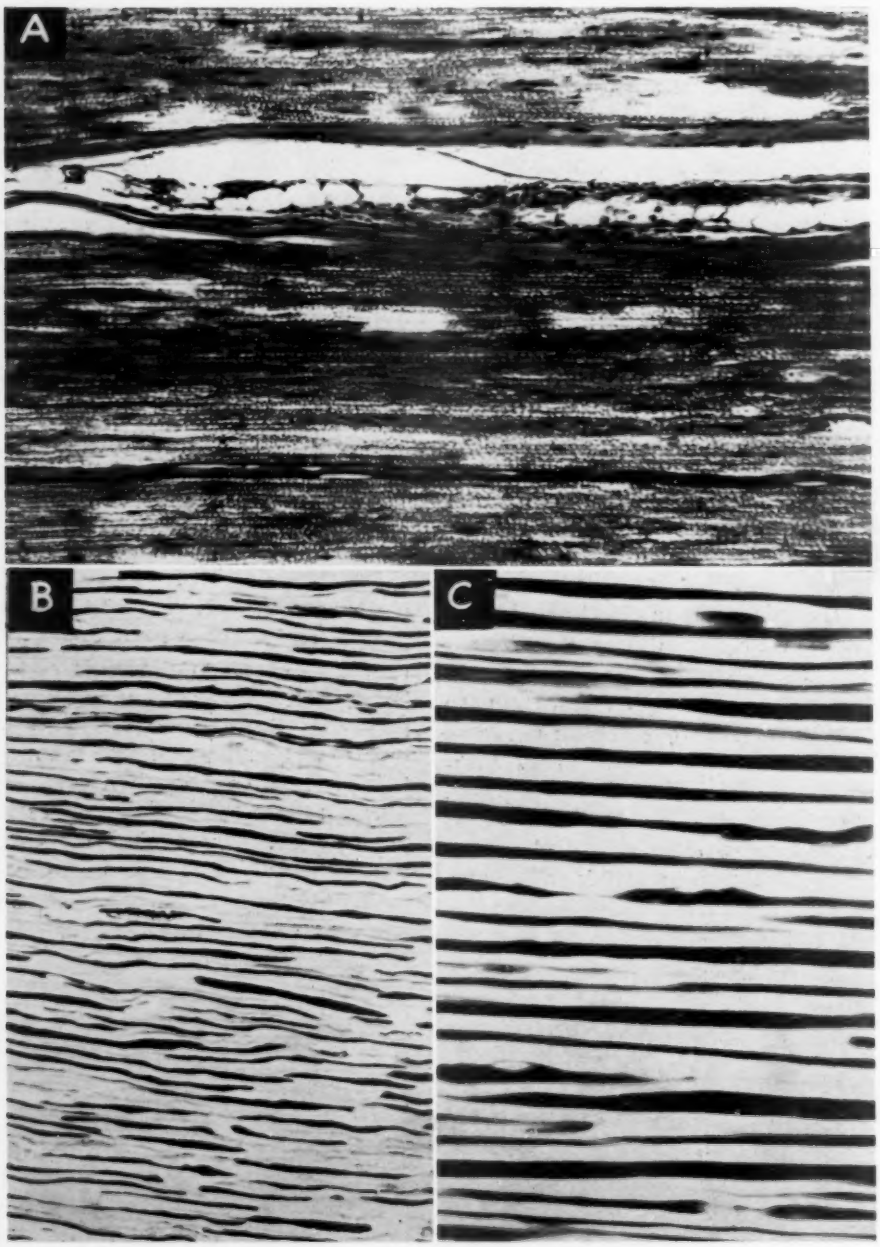

FIG. 5. Photomicrographs (*A*) and (*B*) were taken from sections of sciatic nerves of rabbits which received intraepineurial injections of 0.2 cc. of unaltered pantopaque. Section (*C*) was obtained from the sciatic nerve of a rabbit which received 1 cc. of physically emulsified Pantopaque intraepineurially.

The section shown in (*A*) was taken seven days after operation. The nerve is normal except for very slight monocytic infiltration of the interfascicular connective tissue. This infiltrative change, usually minimal when 0.2 cc. quantities of unaltered pantopaque are used, is considerably intensified with much larger quantities.

Section (*B*) was obtained sixty-seven days after operation. The axis cylinders are normal.

Section (*C*) was taken 182 days after operation. Usually with 1 cc. quantities of pantopaque, such as was used in this rabbit, some axis-cylinder degeneration occurs. Restoration of function and regeneration of axis cylinders apparently occurred in this case.

(*A*) hematoxylin and eosin stain, ×150. (*B*) and (*C*) Gros-Bielschowski impregnations for axis cylinders. (*B*) ×425; (*C*) ×900.

were reversible, however, so that regeneration had occurred by the time the animals were sacrificed.

In 1 rabbit the injection of 1 cc. of pantopaque resulted in loss of the spread reflex, which returned to normal at sixty-five days. This animal was sacrificed 265 days after operation. Adhesions found at the injection site were considerably greater than on the control side. The nerve fibers, however, were normal.

The roentgenographic visualization of the nerves was excellent in these cases.

In 2 rabbits, in which 0.8 cc. and 1 cc. of pantopaque were injected intraepineurially the opaque material entered the thecal sac by way of the lumbosacral nerve roots and passed out from the spinal canal along the opposite lumbosacral nerve roots (Fig. 6).

Chemical emulsions of pantopaque. Since the roentgenographic contrast in nerves injected with pantopaque had been so excellent, it was considered worth while to attempt to modify the compound in the hope of reducing its undesirable effects or facilitating its passage along the nerve. An attempt was made to achieve this objective by emulsifying the pantopaque.

With certain chemical emulsifiers, emulsions of pantopaque and normal saline were prepared immediately before use. The emulsifiers were added to pantopaque and saline mixtures or to pantopaque alone, and then heated until the liquid appeared homogeneous.

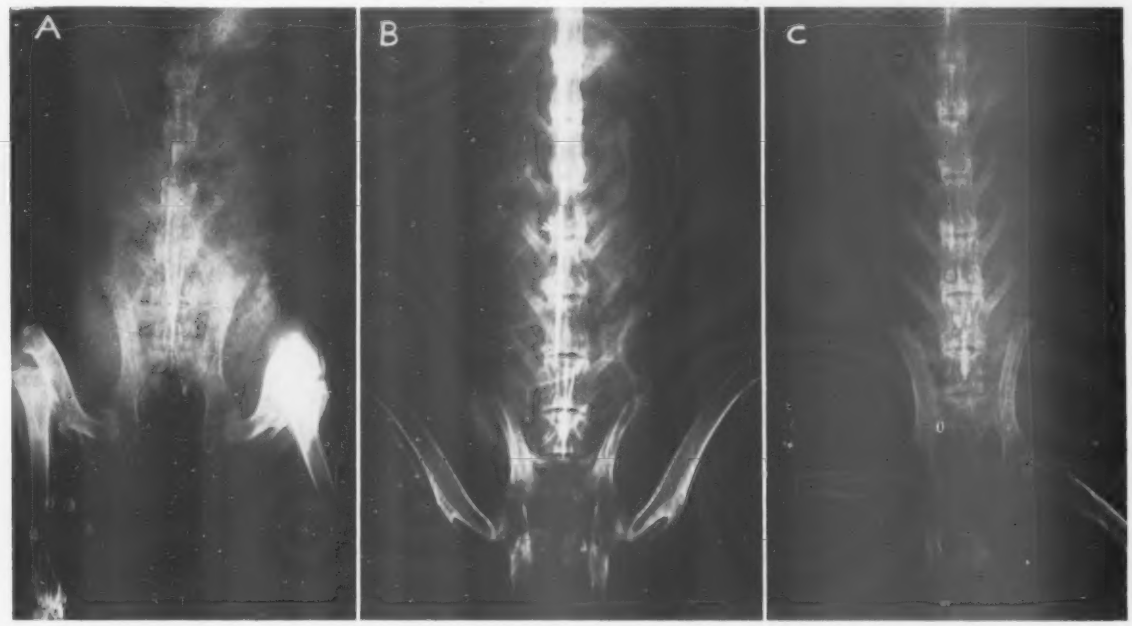

Fig. 6. Roentgenograms taken after the subepineurial injection of 1 cc. of emulsified pantopaque (five parts of pantopaque to one part of normal saline solution emulsified by subjection to supersonic vibration at 1,000 kilocycles per second for three minutes). (*A*) Outlined is the sciatic nerve, in part, as well as two nerve roots and the spinal canal. Note also the emergence of pantopaque along the caudal nerve on the opposite side. This roentgenogram was taken immediately after the injection. (*B*), taken one day later, shows the spinal canal more clearly outlined. (*C*), taken forty-five days after the injection, shows complete absorption of the pantopaque from the injected leg. Some pantopaque remains intraspinally.

I. With Tween 20 and/or 80 (Polyoxyalkylene Sorbitan Monotaurate. Atlas Powder Co., Wilmington, Del.).

Thirteen rabbits received intraepineurial sciatic injections of 0.1 cc. or 0.2 cc. of emulsions* of pantopaque and saline solution. Excellent neurograms resulted, the spinal canal and one or two of the contralateral as well as the ipsilateral nerve roots being outlined in several cases. Absorption of the contrast medium from the nerve occurred within periods varying from three to sixty-six days. Absorption from the spinal canal was much slower (Fig. 6).

Severe functional disturbances followed in all

* The emulsions contained 45 or 50 volumes of pantopaque, 5 or 10 volumes of saline solution and 1 or 15 volumes of one or the other or of both Tweens.

animals. In 1, complete paralysis of both hind limbs occurred immediately after the injection on one side only. Another rabbit developed paralysis of the non-injected hind limb nineteen days after the opposite limb was injected and became paralyzed. The animals survived for intervals varying from four to 110 days.

At necropsy the injected sciatic nerves were either normal or surrounded by adhesions of varying intensity. Rather severe degeneration of nerve fibers occurred in these nerves (Fig. 3*B*).

Emulsions of pantopaque formed by adding Yelkin BSS (approximately 65 per cent vegetable lecithin and 35 per cent soybean oil, Rosso and Rowe, Inc., New York) also produced severe ill effects in the 3 rabbits in which it was tried.

Physical emulsions. Physical emulsions of pantopaque were prepared with a supersonic vibrating machine. Through the courtesy of the Crystal Research Laboratories of Hartford, Connecticut, we were permitted to make up several batches of emulsions formed by vibrations at from 400 to 1000 kilocycles for from thirty seconds to ten minutes. These emulsions contained varied proportions of pantopaque and saline.

There appeared to be no correlation between the functional roentgenographic findings and the vibration frequency at which the emulsion was formed or the time during which the exposure was made.

Seventeen rabbits were used in this series of experiments. The emulsions contained 3, 5, 6, 20, 50 or 100 volumes of pantopaque to 1 or 2 volumes of saline solution. The size of the pantopaque globules varied from 3 mu. to 15 mu.

One rabbit received an intraepineurial injection of 0.2 cc. of emulsified pantopaque. An excellent neurogram was obtained. The animal exhibited no functional or histological ill effects from the injection.

The other 16 rabbits received 1 cc. injections of the emulsified pantopaque. Very good neurograms were obtained in most cases. In about half the cases the material entered the spinal canal by way of one or two of the corresponding nerve roots and emerged from one or two of the contralateral lumbosacral nerve roots. Absorption of the contrast medium from the nerves occurred at intervals varying from forty-five days to four months. In 1 unusual case, in which a good neurogram was obtained, absorption was complete in ten days. Absorption of the pantopaque from the spinal canal was much slower. In 1 case only about 50 per cent absorption from the spinal canal had occurred by the time the rabbit was sacrificed, 182 days after injection.

The rabbits survived for periods varying from eighteen to 182 days. Total paralysis of the injected limb occurred in 6 cases. This persisted in all but one animal. In 1 rabbit the non-injected hind limb was also paralyzed. In this case roentgenograms showed filling of the spinal canal with pantopaque, which had migrated out along two contralateral lumbosacral nerve roots. In 7 animals varying degrees of impairment of spread reflex occurred. In 5 rabbits the reflex returned to normal within a month. There was no detectable functional abnormality in 3 animals.

At autopsy, adhesions, usually minor, were found around some of the injected nerves. Microscopic studies of the injected nerves showed varying degrees of degeneration of nerve fibers, usually slight. Degeneration was somewhat greater in the animals that exhibited paralysis. The epineurial sheaths at the injection sites had thickened and showed slight monocytic infiltration. There proved to be no correlation between the size of the pantopaque globules, on the one hand, and, on the other, the quality of the neurograms, the absorption of the pantopaque, and the functional and histological changes caused by the injection.

DISCUSSION

Our search for a contrast medium for neurography fell short of revealing the ideal. All compounds tested except air, which gave insufficient contrast, lipiodol and pantopaque, resulted in rather severe functional disturbances as well as histo logic abnormalities.

Pantopaque produced satisfactory roentgenographic contrast. It is more readily injected than the very viscous lipiodol. Its innocuousness is not absolute, but quantities of 0.1 cc. or 0.2 cc. injected subepineurially into the sciatic nerves of rabbits usually produced little or no functional or histologic abnormality. With larger amounts of pantopaque ill effects were observed. The effects of pantopaque emul-

sified with physical agents were no different from those produced by ordinary non-emulsified pantopaque.

The functional and microscopic results of neurography vary to a considerable extent with the use of given radiopaque compounds. This is probably due to variations with the precise location of the needle point relative to the nerve fascicles. When the injection mass is confined to the epineurial space, the functional changes are minimal and the microscopic inflammatory changes remain within the epineurial space. It is difficult to limit the injection to this space however. Often the needle point penetrates the perineurial sheath, and the radiopaque material comes to lie within this space or in the parenchyma of the nerve. Degeneration of nerve fibers and consequent functional aberrations then become prominent.

Other studies[22] have demonstrated that severe sciatic paralysis may follow the intraneural injection of penicillin. When the penicillin is deposited in the epineurial space, however, no functional change occurs. The inflammatory reactions in such cases remain confined to the epineurial space.

These experiments have provided abundant evidence that the epineurial and perineurial sheaths protect nerve fibers. This protection, although considerable, is not perfect. Very intense inflammatory reactions in these sheaths may break through the barriers and cause degeneration of nerve fibers.

The functional tests employed in this study were relatively crude, This is indicated to some extent by the lack of correlation between them and the histologic results. Functional derangement as judged by the spread reflex or by observations upon the motor power of the injected limb did not always accompany microscopic abnormality. This is not surprising. The damage to nerve fibers may be too slight to disturb function.

The disappearance of the spread reflex after the intraepineurial injection of a radiopaque compound does not in itself prove the compound deleterious. The reflex occasionally disappeared when the nerve was merely exposed and handled. In such cases histologic studies showed only minimal degenerative and inflammatory changes. In time functional recovery and regeneration followed, with only occasional histologic abnormality, such as slight monocytic infiltration of a somewhat thickened epineurial sheath.

Since elevating a nerve with a nerve hook, though convenient, may result in functional and microscopic changes, it is advisable, whenever possible, to inject the nerve while still in its bed. It is true that holding a nerve taut on a nerve hook does facilitate the injection. However, positioning the neighboring joints to straighten out the tortuosity of the nerve may achieve this effect.

After the attempted intraepineurial injection of the radiopaque compounds used in this study, the loss of spread reflex or other functional alterations was sometimes delayed. This was the case also in experiments with the use of penicillin. It is probably due to the transport of the injected material, so that it comes to lie in intimate relationship with nerve fibers. Or it may be due to increasing inflammation during the first few days or the first week after injection. Such effects, in fact, usually remained latent no more than a week. Occasionally during this period the functional deficit increased gradually.

The injection of emulsified pantopaque into the sciatic nerve of one animal was followed nineteen days later by paralysis of the opposite, non-injected limb. In another animal in which pantopaque, also emulsified with the Tweens, was used, bilateral paralysis of the hind limbs occurred even though the sciatic nerve of but one limb was injected. These results are probably attributable to migration of the emulsified pantopaque into the spinal canal and thence along the contralateral lumbosacral nerve roots to the sciatic nerve.

Such transport of radiopaque compounds has been demonstrated roentgenographically in our study. It occurred in the case of a rabbit into which 1 cc. of physically emulsified pantopaque was injected. The injected sciatic nerve, the subarachnoid space, and two nerve roots of the contralateral lumbosacral plexus were all delineated. The paralysis was probably due to direct toxic action of the emulsified pantopaque on the opposite nerve roots. The spinal cord was unfortunately not obtained for examination in this case. It is possible that a direct toxic effect on the spinal cord, such as we saw[22] in a rabbit after penicillin injection into a nerve, may have occurred in these cases.

Such migrations are understandable in view of the relationship of meninges to peripheral nerve sheaths. The epineurium is the direct prolongation of the dura mater; the perineurium is the peripheral extension of the arachnoid.[21] The epineurial space is therefore potentially connected with the subdural space. Probably when the radiopaque material enters the subarachnoid space, the needle point or the injection mass penetrates the epineurial space and lies within the perineurial space.

There is a space, potentially at least, between the perineurium and the epineurium (epineurial space). Another space exists between the endoneurium—which is the peripheral prolongation of the pia mater over the nerve fibers—and the perineurium (perineurial space). Various connections between these spaces appear to exist. For example, after the injection of thorotrast just under the epineurium, granules of this material may be seen in great numbers adjacent to nerve fibers. It is possible in such cases that the thorotrast was engulfed and transported by the phagocytic cells. Whether such migrations might cross connective tissue septa rather than move along existent, established pathways is problematical.

The question of a true current of fluid within these spaces is unsettled. However, these spaces are injectable. Both centrifugal and centripetal flow occasionally resulted from the same injection. Pantopaque, after being initially injected, showed little or no migration and a very slow rate of absorption over many weeks or months. This fact casts some doubt on the presence of a true fluid circulation within these spaces. The evidence, however, is inconclusive because of the artificial injection pressure used, which might damage nerve tissue. Impairment of the absorptive mechanism might result therefrom.

The delineation of the intraspinal area by the pantopaque injected in our experiments raises the question whether this medium was contained within the subarachnoid or the subdural space or possibly both. French, Strain and Jones[6] have investigated this problem by injecting iodinated compounds colored with carbon to serve as a tracer. The course of this medium was then followed roentgenographically and later by direct microscopic examination. Sometimes it was found to be in the subdural as well as in the subarachnoid space. In the experiments of Sullivan and Mortensen[20] a light brominized oil colored with scharlach R was injected into the sciatic nerve of rabbits. After several hours the colored oil was seen to be present in the subarachnoid space.

There is evidence that active movement of an extremity increases the rate of movement of tissue fluid through it.[4] Therefore, we undertook experiments to determine whether vigorous muscular contractions of an extemity might accelerate the flow of pantopaque injected into the sciatic nerve. Subepineurial injections of 0.2 cc. of pantopaque were made in the sciatic nerves of two dogs and two rabbits bilaterally. After operation, while the animals were still anesthetized, and at daily intervals for the three days following, the muscles of one of the injected lower limbs were stimulated with galvanic current strong enough to produce vigorous flexion contractions at the knee and plantar contractions at the ankle. Approximately fifty such stimuli were applied each day.

There was no evidence in these animals that such stimulations had any effect on the flow of the radiopaque medium in the nerve.

The technique of neurography, whereby the contrast medium is deposited in the epineurial or perineurial space, is at present uncertain and to some extent a matter of chance. If certain precautions are taken, however, satisfactory sciatic neurograms may be obtained in rabbits in at least 50 per cent of the cases. Success increases with practice.

Proper placement of the hypodermic needle is essential for a good neurogram. It must be inserted just through the epineurium and about 1 cm. along the nerve. The medium should be injected so that a small wheal appears and flows freely along the nerve. If extravasation occurs from back pressure or local ballooning without much extension of the material, the needle must be withdrawn. It may then be introduced at another point along the nerve.

After injection with any of the contrast media the sciatic nerve was never completely visualized roentgenographically from spine to heel. Nevertheless, satisfactory visualization of the nerve from spine to knee or below this level often occurred.

The use of pantopaque, or any other contrast medium now available, for delineating peripheral nerves roentgenographically has certainly not reached the stage of development where it can be used as a routine diagnostic procedure. The injection technique is at present too uncertain. It should be reserved for special clinical problems where information concerning the physical status of a nerve and the location of its lesion is urgently required and cannot readily be obtained by any relatively safe procedure. In such cases probably not more than 0.2 cc. of pantopaque should be injected. Larger quantities may seriously damage the nerve. This technique should be used with full awareness of its limitations and only by investigators experienced in its use on experimental animals.

When neurography is attempted, the nerve should always be exposed at operation. Any contrast medium is much better tolerated within the epineurial or perineurial spaces than intraneurially. Some damage to nerve fibers results when the material comes into direct contact with the nerve fibers. For this reason the percutaneous injection of nerves is unsafe.

CONCLUSIONS

1. With intraepineurially injected argyrol, porotargol, hippuran, skiodan, sodium iodide, neo-iopax, 6-iodo-d-galactose, viscorayopake, diodrast or air, either unsatisfactory neurograms were obtained or, when they were satisfactory, the compounds had marked toxic effects upon the sciatic nerve of rabbits functionally and structurally.

2. Thorotrast (6.25–25 per cent) provided satisfactory neurograms. But in view of the known late harmful effects of this radioactive compound, its use for clinical neurography should be discontinued.

3. Very good delineation of the sciatic nerve was obtained with lipiodol. Its intraepineurial injection, however, is difficult because of its great viscosity. Moreover, ill effects were encountered following its injection.

4. Satisfactory neurograms are obtainable with pantopaque, alone or in emulsion form. But—

A. Pantopaque emulsified chemically with Tween 20, Tween 80 or with Yelkin BSS has a markedly deleterious effect on nerves, as functional and histological studies showed.

B. Pantopaque in original form or after emulsification by the supersonic vibrating machine, when injected intraepineurially in amounts of 0.2 cc. in the sciatic nerves of rabbits, produces either no functional or histologic changes or minimal ones. With larger quantities, however, serious functional and histologic abnormalities may occur. These two forms of pantopaque seem to be essentially equal in the quality of the neurograms they produce, in their

absorbability and in their functional and histologic effect on the nerve.

5. The use of small quantities (not more than 0.2 cc.) of unaltered pantopaque for delineating peripheral nerves, although promising in its clinical applicability, cannot yet qualify as a routine diagnostic procedure. The search for more suitable contrast media should be continued.

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ISODOSE MEASUREMENTS OF LINEAR RADIUM SOURCES IN AIR AND WATER BY MEANS OF AN AUTOMATIC ISODOSE RECORDER*†

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INTRODUCTION

THE Paterson and Parker gamma-ray dosage system is constructed mathematically on one confirmed calibration: 1 milligram hour exposure from a point source of radium filtered with 0.5 mm. platinum, delivers at a distance of 1 cm. an intensity of 8.4 gamma roentgens. From this value, dosage tables were constructed mathematically for simple and complex arrangements of radium sources. The data in these tables have not yet been satisfactorily confirmed by measurements.

For a simple linear radium source, the calculated intensity distribution for the gamma radiation coming from the sides is probably correct. The radiation field around the ends, however, can be obtained mathematically only with some approximations[8,10] due to the unknown self-filtration factor of radium and the complexity of mathematical evaluation of oblique filtration through the walls of the capsule. With the aid of a scintillation counter as a gamma-ray detector, together with an automatic isodose recording device, the radiation fields around various radium sources were surveyed by means of isodose curves and compared with those derived mathematically.

CLINICAL NEEDS FOR IMPROVED ISODOSE STUDIES

A biological demonstration of the distribution of the radiation around a tandem of linear radium sources is shown in Figure 1. A formerly employed technique for treating keloids entailed the placement of four radium tubes on an abdominal scar immediately after excision of a keloid in a female Negro. Each tube contained 10 mg. radium with an active length of 2 cm. and filtration of 1.5 mm. Pt; exposure 240 mg. hr. per tube. An intense second degree

FIG 1. Second degree skin reaction of four radium tubes in tandem on an abdominal scar of a female Negro after excision of a keloid.

skin reaction, measuring 2 cm. in diameter was obtained in ten days (Fig. 1). This reaction was more intense near the center of each radium tube and less intense around its end. The region of obviously low radiation effect, where the ends of two tubes were touching each other, is remarkable. Published isodose curves do not portray isodose patterns quite consistent with this finding.

Various radium techniques are applied in treating carcinoma of the corpus uteri. Dosage estimation following insertion of

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several tandems or multiple tubes, whose long axis is perpendicular to the fundus, requires an accurate knowledge of the radiation distribution at the ends of the sources. Where a triangular arrangement of linear radium sources is employed, the problems are quite similar. Radioautographs (Fig. 2) indicate that the radiation

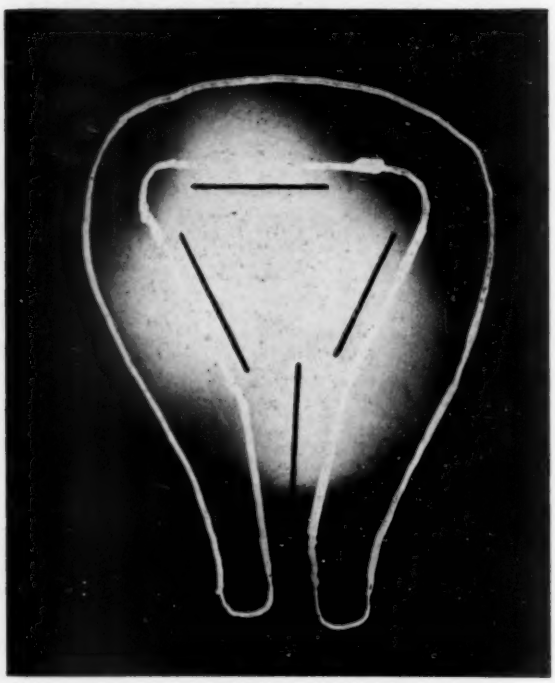

FIG. 2. Radioautograph of a triangular arrangement of four radium tubes for treatment of carcinoma of the corpus uteri.

field at the cornu where two tubes are pointing is weak.[2] A more exact knowledge of the distribution of radiation coming off the ends of a radium capsule would be helpful.

In the Walter Reed technique for treating cancer of the bladder, an inflatable balloon catheter containing a single radium tube is inserted into the bladder, and the long axis of the radium capsule points toward the vesical neck.[3] Since most bladder cancers are located in the lower portion of the bladder around the vesical neck and trigone, they are irradiated predominantly by radiation coming off the ends of a radium capsule. It is therefore necessary to have a precise knowledge of the intensity distribution around these gamma-ray sources.

Similarly, one of the techniques used in the treatment of cancer of the maxillary antrum entails placing a single radium capsule in the center of the antral cavity. Improper understanding of the radiation field has been responsible for some failures.

The above examples, which are only a few of a great number, illustrate the need for an accurate knowledge of the radiation field around radioactive sources employed in radiotherapy.

THE AUTOMATIC ISODOSE RECORDER

An instrument[5] has been developed recently which enables the experimental investigation of the radiation field of a single source as well as of a number of gamma-ray sources arranged in any configuration. The complete results are obtained in a very short time and with a high accuracy compared with other methods. The main features of the instrument will be discussed here in a somewhat less technical form than described previously in a physical journal.[5]

The gamma-ray detector used in this instrument is a scintillation counter which overcomes some of the defects of the air ionization chamber when employed for this purpose.[6] The radiosensitive part of the scintillation counter consists of a small, cylindrical, calcium tungstate crystal 1/8 inch in diameter and 1/8 inch in height (volume 0.03 cm.^{3}). The incident gamma rays produce small light flashes in the crystal which are conducted through a long quartz rod to a light-sensitive tube (photomultiplier). This tube converts the light flashes into electric pulses which can be amplified and transmitted through cables to the recording part of the instrument.

The isodose recorder is built in two units, both partly visible in Figures 3 and 4. The two assemblies can be positioned at a distance from each other so that the radiation intensity at the recording device always

remains within safe limits.

The photomultiplier tube, covered by an aluminum light shield, is situated above the water tank visible in Figure 3. The thin aluminum tube pointing down into the water tank is connected with the photomultiplier housing. It contains the light-conducting quartz rod (15 inches long) and the scintillation crystal mounted at the bottom end of the rod. The radium source, placed near the crystal at the center of the water tank, is mounted on a vertical motor shaft protruding up through the floor of the tank, which can be filled with water as a scattering medium.

When tracing an isodose curve, the radium source is rotated slowly at two to six minutes per revolution. The scintillation crystal, controlled by an electronic circuit, can automatically slide in a straight line toward and away from the radium source. For tracing an isodose curve, the electronic circuit is preset to accept a fixed number of scintillations per second (e.g., corresponding to 50 roentgens per hour). During the course of a single rotation of the radium source, the counter scans the field of radiation emitted from the source. It automatically moves to a position relative to the source at which it will continually register that number of scintillations which is equivalent to the preset value (e.g., 50 r/hr.).

In the recording apparatus (Fig. 4) the relative movements of the source and counter are duplicated. The turntable with recording paper rotates synchronously with the gamma-ray source. The recording pen moves back and forth simultaneously with the scintillation counter. This movement is accomplished by a servomechanism. As a result, the pen draws an isodose curve on the paper while the instrument is in operation.

By selecting, through the electronic circuit, various intensity values, a set of isodose curves is traced in the same plane. The source may then be raised or lowered and a new corresponding set of curves recorded in a different selected plane.

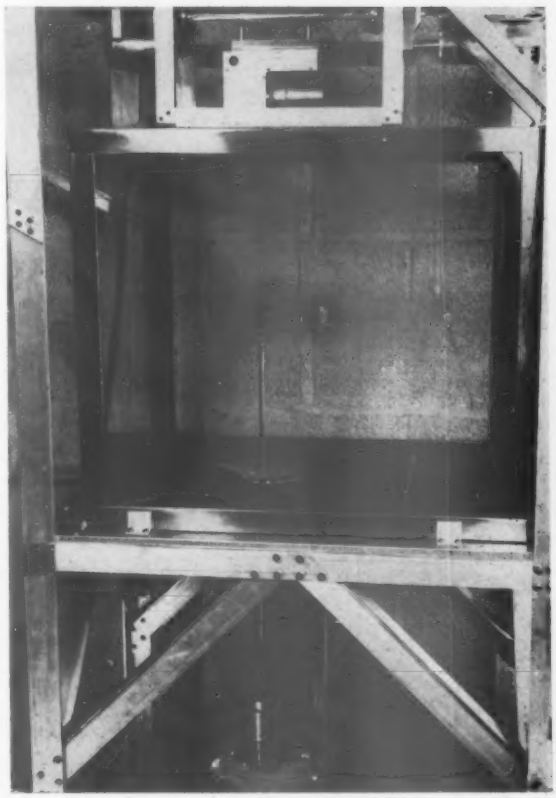

FIG. 3. Scintillation counter assembly. In the center of the water tank are the radioactive source and the radiation detector.

CALIBRATION OF THE SCINTILLATION COUNTER

The measurements of radiation doses in roentgens requires the determination of the number of ion pairs produced in a given volume of air. The ionization chamber is the only instrument known which allows the performance of such measurements.[1] Other radiation detectors such as the Geiger-Müller counter or the scintillation counter register only a fraction of the incident gamma rays.[4] The number of counts registered is not an indication of the energy spectrum of the radiation, and dosages cannot be determined from the number of gamma rays present in a given volume if their energies, or that fraction of their energies absorbed in the volume, is unknown. Therefore, the scintillation counter in its present form is calibrated by

comparison with ionization chamber measurements.

As mentioned above, the intensity of a 1 mg. radium point source filtered with 0.5 mm. Pt is known to be 8.4 r/hr. at 1 cm. distance.[1] The attenuation of the radiation intensity is given by the inverse square law for distances within which the absorption of the radium gamma rays in air can be neglected. Therefore, the radiation intensity of a point source can easily be calculated and expressed in roentgens.

By traversing the field of a calibrated radium source with the scintillation counter, the relation between the number of registered counts and the dose in roentgens as a function of distance from the source center can be established. A calibration obtained in this manner is limited to the recording of isodose curves surrounding radium sources. Similar calibration curves from other emitters are required because of the difference in the gamma-ray spectrum from different emitters.

For mediums other than air, the gamma-ray distribution around a point source due to scattering of the gamma rays is not known. Therefore, the radiation field of a point source in a scattering medium cannot be calculated by the inverse square law. Calibration is possible, however, by comparing the number of counts registered at various distances from a point source in a scattering medium (water) with ionization chamber measurements taken under the same conditions.

FIG. 4. Recording assembly. On the top plate of the electronic cabinet are shown turntable and recording pen.

COMPARISON OF RADIUM TUBES OF DIFFERENT CONSTRUCTION

Radioautographs (similar to the one in Fig. 5) of different types of radium tubes show that the radiation fields around both ends of each tube vary considerably.[2] There is a broad conical zone of markedly diminished intensity at each end. Traversing one or both of these zones (depending on the amount of platinum filtration) is a pencil beam of relatively increased intensity. The diminished intensity is due predominantly to heavy oblique filtration. The pencil beam appears at that end of the tube where the filtration is approximately

that of the stated wall thickness (e.g., 0.5 mm. Pt). At the opposite end, where the tube has been finally sealed by a thick plug of platinum, the filtration is higher, and the radiation intensity is reduced. Some tubes have an eyelet in the latter end, but this does not significantly change the filtration.

Four different types of linear radium tubes have been investigated with the automatic isodose recorder. The specifications of these tubes are listed as A, B, C, and D in Table 1.

Three individual tubes of Type A were tested for uniformity of their construction. They yielded the same isodose curves within the accuracy of the instrument. In Figure 6 a portion of one recording of isodose curves is compared with mathematically constructed curves, derived from data in the tables of Wolf[9] and Quimby.[7] These tables do not take into account irregularities due to construction characteristics of the two ends of the radium tube. The mathematically derived isodose curves are consequently symmetrical with respect to the perpendicular central axis. The experimentally obtained isodose curves agree well with the calculated field distribution around the sides of the radium source. However, near the tube end with the heavier filtration the isodose recorder shows the radiation intensity to be significantly reduced.

By rotating the obtained iosdose pattern around the tube axis, the complete three-dimensional isodose surfaces can be constructed. Close to the radium tube they have the shape of a prolate spheroid (egg-shaped), as shown in Figure 6. Beyond a distance of 2 cm. from the central axis of the source under consideration, the isodose surfaces gradually assume the shape of an oblate spheroid (tangerine-shaped), in which the radium source is eccentrically situated between the two poles. This is clinically significant in intracavity irradiation with a single centrally located radium source.

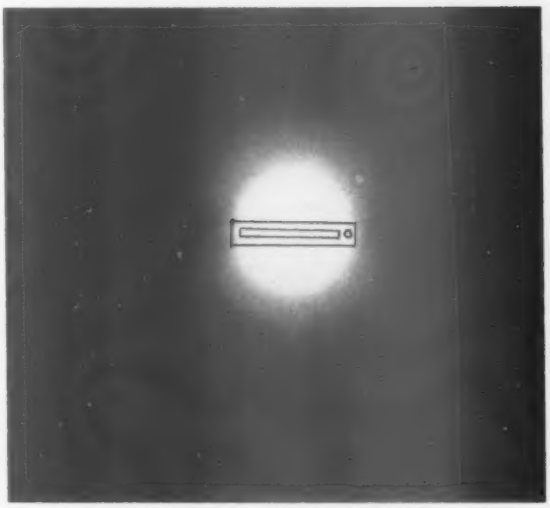

Fig. 5. Radioautograph of a single radium tube (0.5 mm. Pt filtration).

Source B with 1 mm. Pt filtration produces the same general shape of isodose curves (Fig. 7) as source A with 0.5 mm. Pt filtration. Its intensity is reduced as expected.

Table I

	Quantity of Radium (mg.)	Active Length (mm.)	Filtration (mm. Pt)	External Diameter (mm.)	External Length (mm.)	Type of Construction
A	25	12	0.5	2.25	15	No eyelet
B	25	10	1.0	3.5	15	Round end with eyelet
C	26.4	10	1.5	6.5	18	Capsule (wall thickness 1.3 mm. platinum) containing eight 3.3 mg. cells (wall thickness of each cell 0.2 mm. Pt). One tapered end with eyelet
D	50	20	1.5	4.5	25.5	Round end with small eyelet

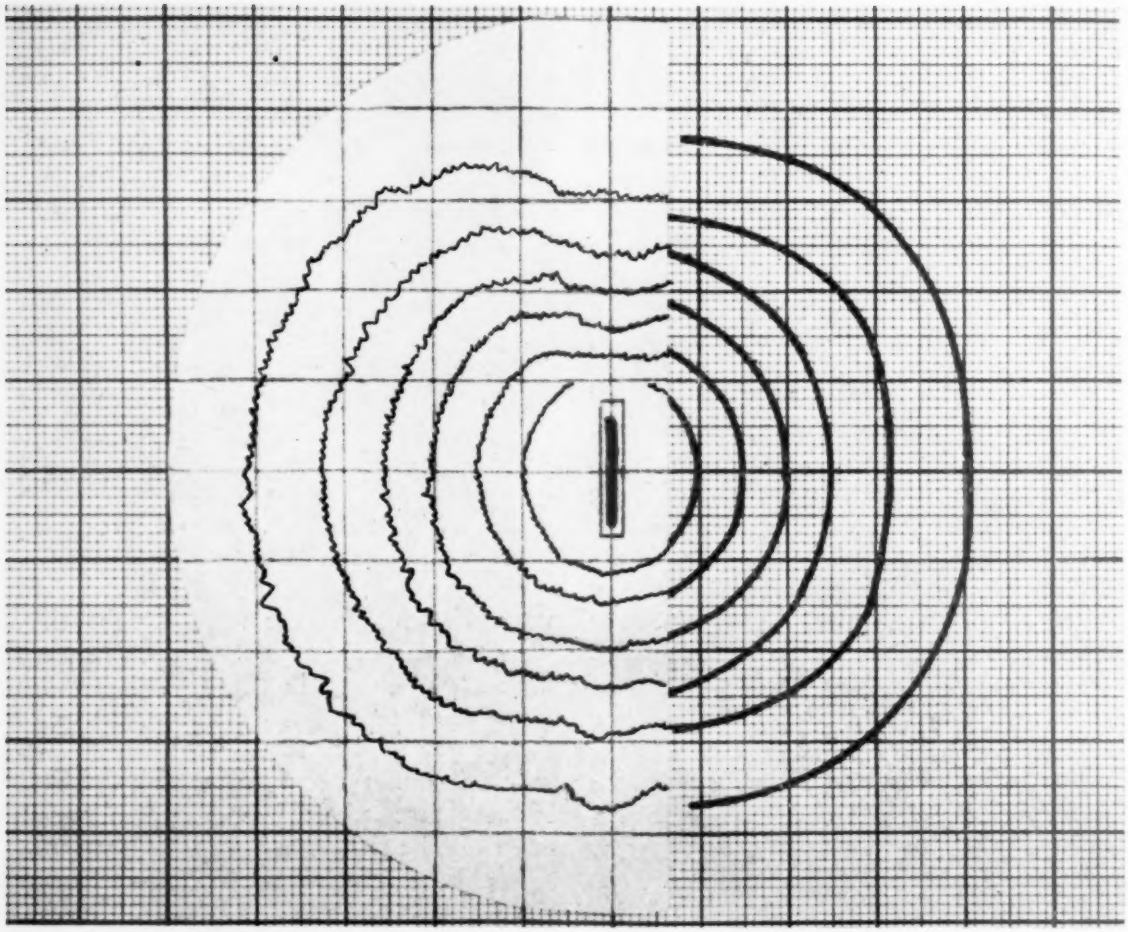

FIG. 6. Calculated and recorded isodose curves of a 25 mg. radium tube, 0.5 mm. Pt filtration. Intensities are 180, 90, 50, 33, 22, and 13 roentgens per hour.

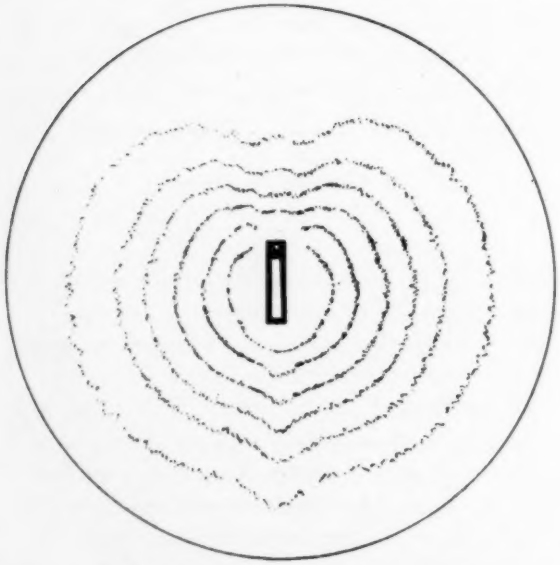

FIG. 7. A 25 mg. radium tube with 1 mm. Pt filtration (180, 90, 50, 33, 22, and 13 roentgens per hour).

FIG. 8. A 1.3 mm. Pt capsule containing 8 cells 3.3 mg. radium and 0.2 mm. Pt each. Isodose intensities 180, 90, 50, 33, 22, and 13 roentgens per hour.

Source C, of completely different interior construction (see Table 1), produces isodose contours with pronounced flattening at the poles (Fig. 8). Its complex construction has made it impossible to calculate isodose curves accurately. The automatic isodose recorder discloses the shape of the curves from such a source in a short period of time.

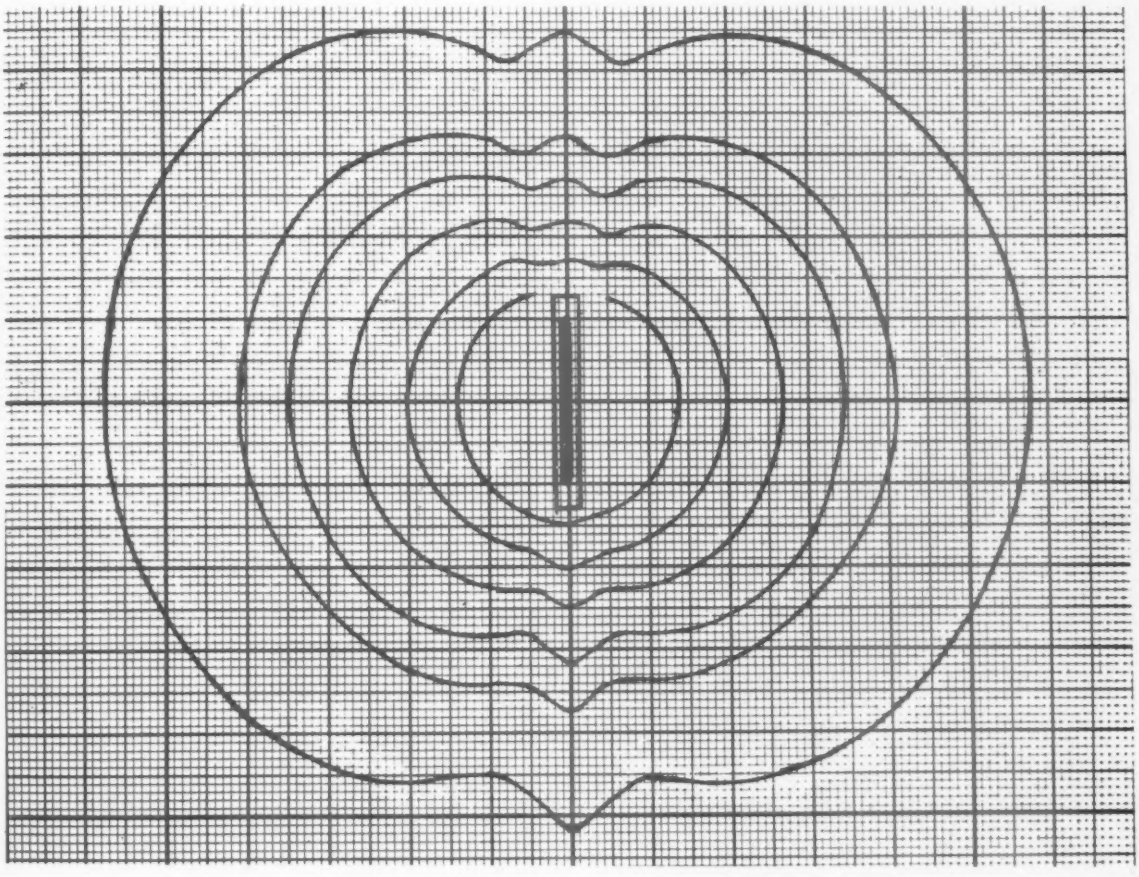

Fig. 9. Drawing of recorded isodose curves for a 50 mg. radium tube with 1.5 mm. Pt filtration. (Intensities 180, 90, 50, 33, 22, and 13 roentgens per hour.)

Source D, with a heavy filtration similar to that of source C, also shows isodose contour shapes of oblate spheroids (Fig. 9). A filtration of 1.5 mm. Pt is advantageous for an improved tube construction producing more nearly symmetrical radiation fields at the ends.

The dimensions of the scintillation crystal (diameter 1/8 inch, height 1/8 inch) limit the resolving power of the instrument. At any point in the radiation field, an average radiation intensity over the crystal volume only can be determined. In regions of slowly changing radiation intensity the present resolving power is adequate. However, at the tube ends, where the pencil beam has a width comparable with the dimensions of the crystal, the results are strongly influenced by the finite resolving power (Fig. 6, 7, and 9). The use of an even smaller crystal would improve the resolution considerably and would define the pencil beam more clearly.

Not included in Table 1 were several tubes wherein it was suspected that the radium salt was improperly packed within the capsules. The isodose curves of two out of three 15 mg. sources (0.5 mm. Pt filtration) with the same specifications were found to be identical and symmetrical

with respect to their perpendicular center axis. The third tube, however, showed a distorted pattern with an increased intensity at one part of the tube. This picture could not be changed by striking the tube rather hard in order to shake the radium inside. A considerable fraction of the radon emanated by the radium adheres to the latter, and the gas is not uniformly dispersed throughout the tube. A defect of this type can be detected with isodose recording.

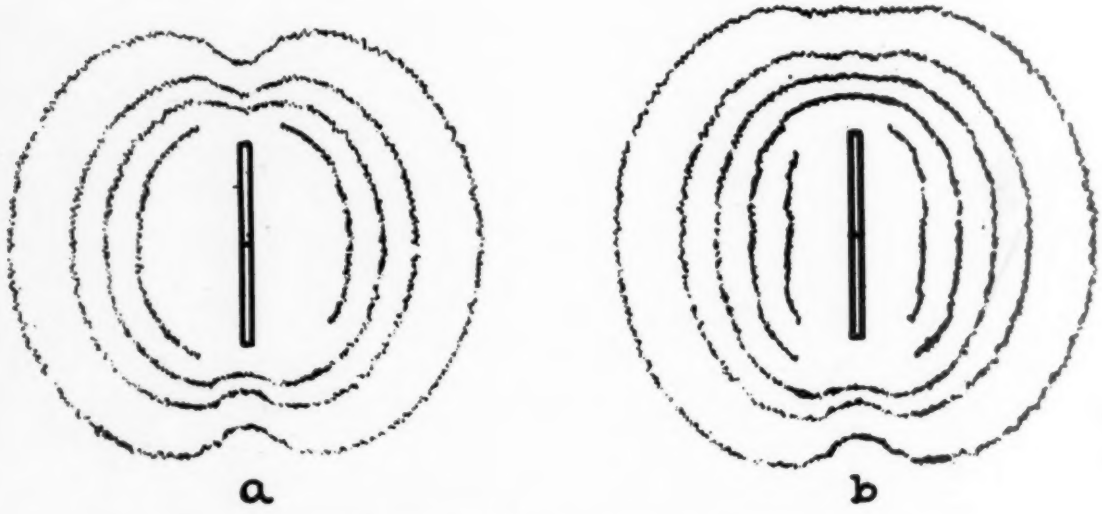

FIG. 10. Two 25 mg. radium tubes in tandem (0.5 mm. Pt filtration). The ends of heavy filtration are pointing (*a*) at opposite directions; (*b*) at the same direction.

ISODOSE CONTOURS PRODUCED BY MULTIPLE SOURCES

Since simple linear sources produce asymmetrical isodose curves, multiple linear sources in tandem arrangement should yield results dependent on the position of the tubes relative to each other. Figure 10 shows two sets of isodose curves from the same pair of radium tubes. In Figure 10*a* the plugged ends of the tubes point in opposite directions. A symmetrical pattern with indented ends is produced. In Figure 10*b* the upper tube is inverted so that the plugged ends point in the same direction. The expected asymmetrical pattern appears. When the plugged ends are placed in contact, the resultant isodose contours are again symmetrical, but less indented at the poles.

Figure 11 was recorded from a tandem composed of four radium tubes; two tubes contained 25 mg. radium each and two 12.5 mg. each. Filtration was 0.5 mm. Pt for each tube. Two sets of isodose curves of corresponding intensities were recorded. Figure 11*a* shows the distribution in the central plane of the four sources, and Figure 11*b* in a parallel plane 7 mm. above the central plane. By this method three-dimensional isodose surfaces can be constructed for any combination of sources.

COMPARISON OF AIR AND WATER MEASUREMENTS

So far only preliminary studies have been made to investigate the effect of a scattering medium surrounding the radium source and the gamma-ray detector. A few results will be indicated briefly since they show the necessity for more thorough investigation.

In the region close to the radium source the presence of a scattering medium (water) does not change the intensity distribution significantly. However, at some distance

from the source (about 3 cm. from a 1.5 cm. length tube), an increase in intensity seems to appear in comparison with the air measurements.*

By raising the water level in the tank (Fig. 3) while the source and the scintillation crystal remain at fixed positions, the following observations were made. As the water level was being raised slowly in the tank, an increase in intensity became visible when the water was 3 inches below the plane of the source and scintillation crystal. The dose increased until the water level reached the plane of the source and scintillation crystal. As soon as the water entered the region between source and detector, a sudden decrease in intensity was observed. This is due to absorption and deflection of a portion of the gamma rays by the water. A further rise of the water level again produced an increase of the measured intensity until the water reached a level about 3 inches above the source plane. Scattered gamma rays, which had not previously, now did reach the detector, since the scattering in air is negligible in comparison to that in water.

The shape of the isodose curves, as expected, is almost the same in air and water.

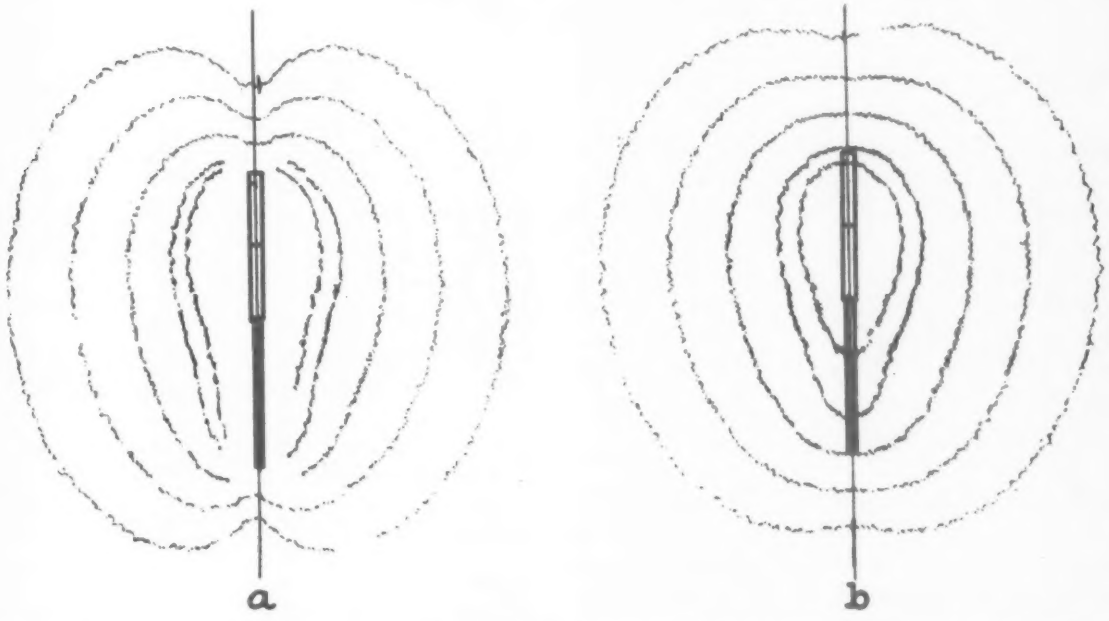

Fig. 11 Tandem of four radium tubes (2×12.5 mg. and 2×25 mg. with 0.5 mm. Pt filtration each). Intensities 250, 500, 1,000, 2,000, and 3,000 roentgens per 1,000 mg. hr. (*a*) Isodoses in center plane of the sources. (*b*) Corresponding isodoses in a parallel plane 7 mm. above the center plane.

However, the dose increases significantly due to the scattered gamma rays. The exact percentage increase has not yet been determined, but is definitely more than 10 per cent in some instances, and not negligible as has frequently been assumed.

DISCUSSION

The tracing of isodose curves described so far for radium sources can be applied to any other gamma-ray emitter. With the present instrument, isodose curves are recorded in a short period of time (two to six minutes). The sensitivity of the apparatus is adjustable to radiation intensities between 10 and 200 roentgens per hour. However, by changing the pulse

* By surrounding the scintillation counter with 1 cm. lucite, the measurements in air or water were not affected. This indicates that under the present conditions[5] soft beta rays do not contribute to the detected intensity.

amplification, this dose interval can be extended in both directions. The accuracy of the recording is limited to about ±5 per cent by the stability of the electronic circuits and the response of the servomechanism. Both the speed of the isodose tracing and the accuracy obtained are adequate for practical clinical requirements. This is an improvement over previous methods.

The scintillation crystal used in this first experiment is smaller than available air ionization chambers. Nevertheless, the crystal could not trace curves closer than 5 mm. from the radium tube. A smaller crystal can easily be employed. It would permit survey of the radiation field closer to the radium tube. It would also increase the detector resolution so that narrow deviations, like the pencil beam coming from the end of the tube, could be more accurately delineated.

SUMMARY

By means of an automatic isodose recorder, which employs a sintillation crystal as a radiation detector, isodose curves around radium sources can be measured in a short period of time with a good degree of accuracy. With certain exceptions, the isodose curves obtained with this instrument conform with isodose curves constructed mathematically. Commonly employed radium tubes show an asymmetrical isodose pattern, rather than the symmetrical pattern described by numerous authors. Increasing the filtration of a radium source reduces the intensity, as expected, and flattens the isodose curves at the tube ends. The presence of a scattering medium (water) surrounding the radium source, raises the dose at points not too close to the source by at least 10 per cent.

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CLINICAL ISODOSE CURVES*†

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INTRODUCTION

THE present paper is of the nature of a progress report on a systematic investigation into the nature of isodose curves in finite phantoms. The bulk of the depth dose data available to clinicians has been obtained by measurements along the central axis of a roentgen-ray beam, in an infinite, homogeneous, tissue-equivalent phantom. Clinical depth doses may differ from these conventional values due either to the finite size or unusual shape of the irradiated region, or to the presence of bone or fat. The work being reported here indicates that it is possible to make some useful generalizations which serve to indicate the nature and extent of the deviations to be expected from the conventional, central-axis determinations and which serve also to simplify the determination of isodose curves in finite phantoms.

The measurements to be reported were made with a 200 kv. (peak) roentgen-ray generator using a Villard circuit, at 50 cm. focus-skin distance, with 0.4 mm. Cu plus 1 mm. Al added filter, giving a half-value layer of 1 mm. Cu. The fields were defined by lead-lined cones, with the limiting aperture at the surface of the phantom.

INSTRUMENTATION

Water has been used in this investigation as the phantom material. It has the following advantages: (1) its effective atomic number and density are close to that of tissue; (2) it is easily secured, in the same form, by all experimenters; (3) the placing of ionization chambers at arbitrary positions is relatively simple; and (4) it allows simple alignment techniques, combining visual sighting with use of a roentgenoscopic screen. There are two disadvantages to water as a phantom material: It requires water-tight ionization chambers; and the chambers can be introduced only from the top, requiring a horizontal roentgen-ray. It is believed that these disadvantages are outweighed by the advantages.

A condenser ionization chamber should have the following properties to be suitable for phantom measurements in water: (1) its active volume should be small in size. so that the dosage distribution is not appreciably disturbed, and measurements are meaningful in regions of appreciable dosage gradient; (2) the walls of the measuring volume should be sufficiently air-equivalent to give isodose curves valid for dosage calculations in tissue; (3) it must be of sufficient sensitivity and stability; (4) it must be waterproof and reasonably rugged; and (5) that part of the chamber which is in the primary beam should be of such size, density, and atomic number that simultaneous measurements with multiple chambers is feasible. Commercially available thimble chambers are not considered satisfactory. In Figure 1 is shown a condenser chamber which at least approximately meets these specifications, and which is made of inexpensive and easily available materials. The stem is $\frac{1}{4}$ inch diameter polystyrene tubing. The central electrode is magnesium. The cap over the measuring volume is wood. The assembly is waterproofed by dipping in collodion, or by covering with a thin rubber sheath. These chambers have the disadvantage that there is a small, apparently radiation-induced leakage, originating in the polystyrene, which leads to an uncertainty of a few per cent of full scale. This can be corrected for with satisfactory accuracy.

The electrical instrument used in charging and measuring the discharge of the

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condenser chambers has been the Victoreen minometer electrometer. This instrument has the advantage of being simple, stable, and commercially available, and is of adequate precision.

The chambers have a full scale sensitivity of approximately 40 roentgens. Checked against the Victoreen r meter, in air, they show a variation in sensitivity of 8 per cent between 4 mm. Cu half-value layer at 400 kv. (peak) and 0.5 mm. Cu half-value layer at 140 kv. (peak).

These condenser chambers and the techniques by which they are used, have been

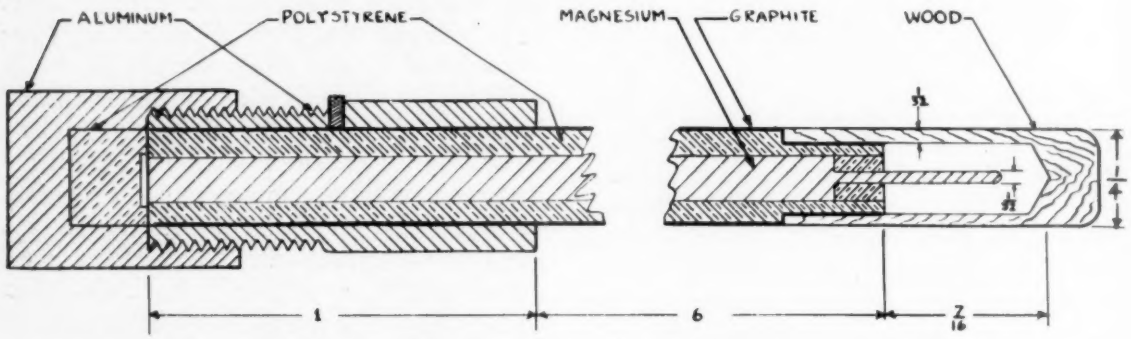

FIG. 1. Schematic cross-section of condenser ionization chambers. Dimensions are in inches. The cap at the left is removed to make contact with minometer electrometer. The chambers are waterproofed by dipping in collodion.

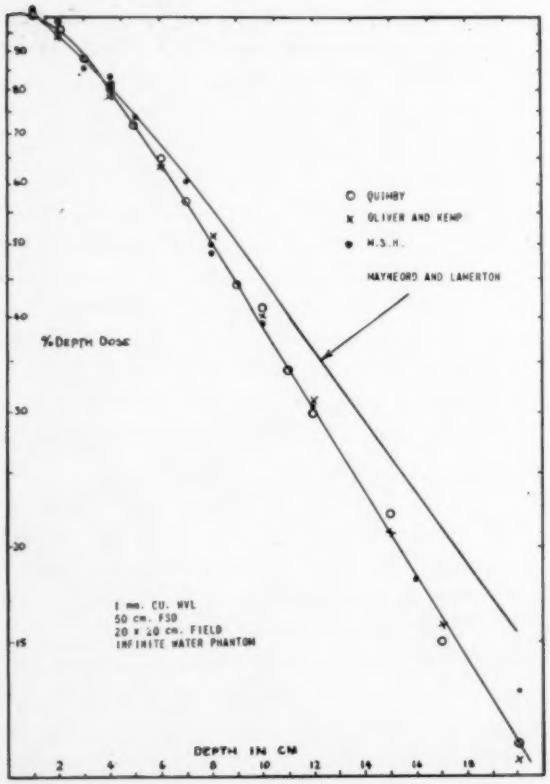

FIG. 2. Central axis, infinite phantom depth dose measurements made in the present investigation, compared with published data.

checked by comparing depth dose measurements made with them against accepted, published values. A typical comparison is shown in Figure 2. Our data agree well with the extrapolation chamber-pressdwood data of Quimby,[3] and the water phantom-condenser chamber data of Oliver and Kemp.[2] The Mayneord and Lamerton curve, taken from their survey of published data,[1] is not in good agreement, for reasons discussed by Oliver and Kemp.

During the development of the chambers it was necessary to know how nearly air-equivalent the cap over the measuring volume must be to give valid depth dose measurements. In Figure 3 is shown a comparison of depth dose curves made with ionization chambers of different materials. The relative depth dose measurements are essentially the same, whether the measuring volume cap is made of aluminum (atomic number 13), or carbon (atomic number 6), or wood (effective atomic number about 7), even though the responses of these three chambers to beams of different energy, in air, showed strong wavelength differences. It can be concluded that depth dose meas-

urements, at least for roentgen-ray beams in the region of 1 mm. Cu half-value layer, are not sensitive to the atomic number of the ionization chamber material. The effective atomic number of wood, just under 7, is closer to that of air, which is about 7.3, than any other simple material.[4]

INFINITE PHANTOM MEASUREMENTS

The word *infinite*, when applied to a roentgen-ray phantom, means "so large that larger will not change the readings." To determine how big a phantom must be in order to be infinite in effect, it is necessary to determine experimentally how much phantom material is needed to saturate the scattering effect at the points where readings are taken. Our results, in agreement with published data by Quimby,[3] indicate that for saturation about 15 cm.

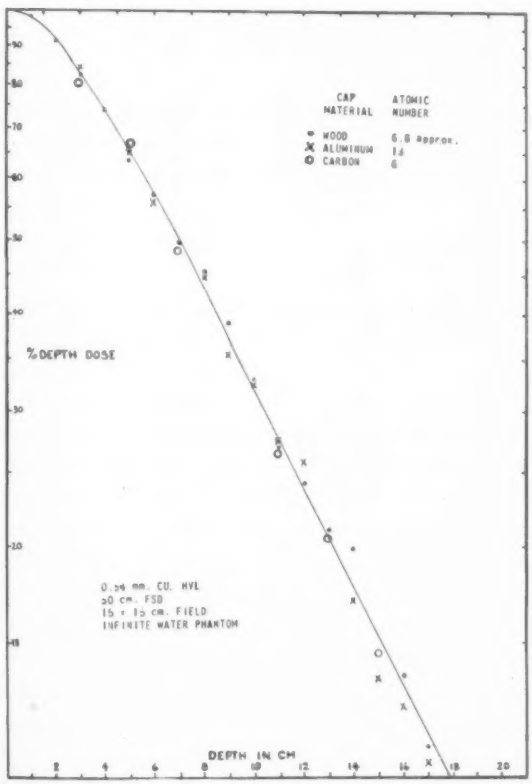

Fig. 3. Comparison of depth dose measurements made with ionization chambers of three different materials. The percentage depth dose is not a function of the wavelength response of the ionization chambers, within reasonable limits.

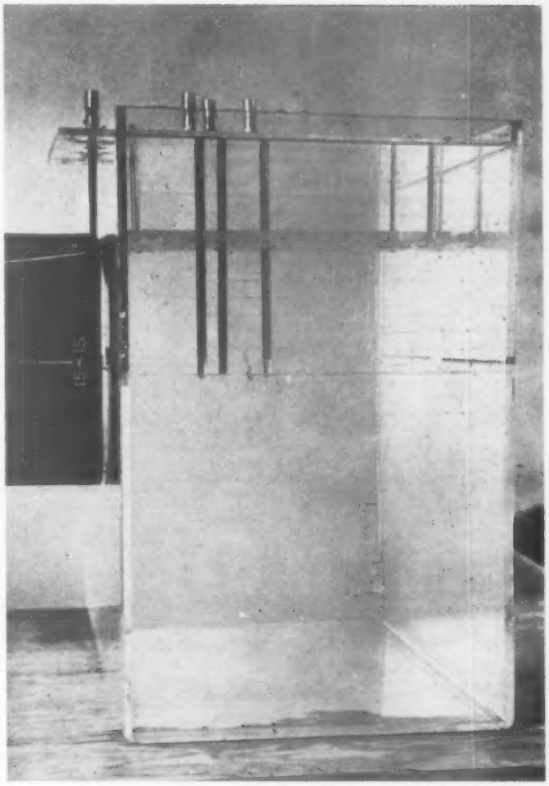

Fig. 4. The infinite water phantom, with four chambers and a treatment cone in place. The water level is about 2 cm. from the top.

of phantom material is required distal to the point of measurement, and that 1 or 2 cm. are required outside the primary beam. This statement, of course, applies to a radiation quality of 1 mm. Cu half-value layer.

In Figure 4 is shown the plastic tank used for the infinite phantom measurements. The tank walls are $\frac{1}{4}$ inch acrylic plastic. It is considerably larger than the beam in cross-section and 25 cm. deep in the direction of the beam axis. It has been backed by 15 cm. of pressdwood, not shown in the figure, placed on the distal side, to give infinite phantom measurements to a depth of 20 cm. It was demonstrated that several chambers could be used simultaneously provided that their shadows did not overlap. The chambers are supported by a plastic slab at the top of the phantom, in which a rectangular array of holes at 1 cm.

spacing has been drilled. The alignment of the chambers is accomplished by a second plastic slab, with an array of holes exactly matching the top slab, mounted about 6 cm. below the top of the tank. The apparent displacement of the aluminum bases of the chambers from the plastic stems is due to refraction by the water. One chamber is half-imbedded in the surface, to measure

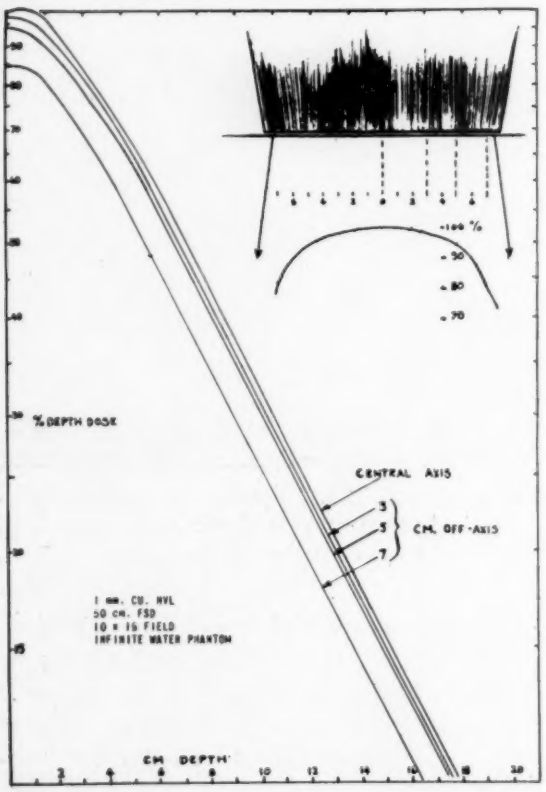

FIG. 5. Central axis and off-axis depth dose curves in an infinite phantom. At the upper right is a schematic of the experimental set-up, with a transverse fall-off curve. The transverse fall-off is taken as 100 per cent on the central axis, and is the same at all depths, within the primary beam.

the surface dose, though it is not apparent in the figure.

Using this infinite phantom, depth dose measurements were made both on and off the central axis, with rectangular and circular fields of various sizes. Typical results are shown in Figure 5, where the percentage depth dose is shown plotted against the depth in centimeters, for a 10 by 15 cm. field. Depth dose measurements have been made along the central axis, and along lines 3, 5, and 7 cm. off the central axis and parallel to it. The sketch in the upper right-hand corner shows diagrammatically the experimental set-up, with the cone at the surface of the phantom, the diverging primary beam, and the lines of measurement shown by broken lines. There are three things to be noted in this figure: First, it is a semi-logarithmic plot, and, as is well known, a straight line on such a graph means exponential attenuation. Second, the central axis depth dose curve is accurately a straight line for depths greater than about 4 cm. (This is also demonstrated by the published depth dose data cited above over a wide range of quality.) Third, the off-axis depth dose curves have the same general shape as the central axis curve, and beyond 4 cm. are accurately parallel to it.

At first sight it might not seem surprising that the attenuation is exponential beyond the first few centimeters, but it must be remembered that the beam consists of a continuously changing mixture of primary and secondary radiation. At present, it is not possible to explain theoretically this exponential attenuation. It must be considered an experimental observation, useful because it simplifies calculation of depth doses.

The fact that the off-axis curves are parallel to the central axis depth dose curve has practical significance. It means that the depth dose curve along any line parallel to the axis may be obtained by multiplying the central axis curve by a constant. Moreover, the value of this constant is not a function of the depth, but only of the distance from the central axis. These constants can then be determined by measuring the fall-off from the central axis dose along a line perpendicular to the axis, at a single convenient depth. For example, in the 10 by 15 cm. field illustrated, the measurements perpendicular to the axis were made at a depth of 5 cm. In this case, the transverse fall-off curve, which is taken

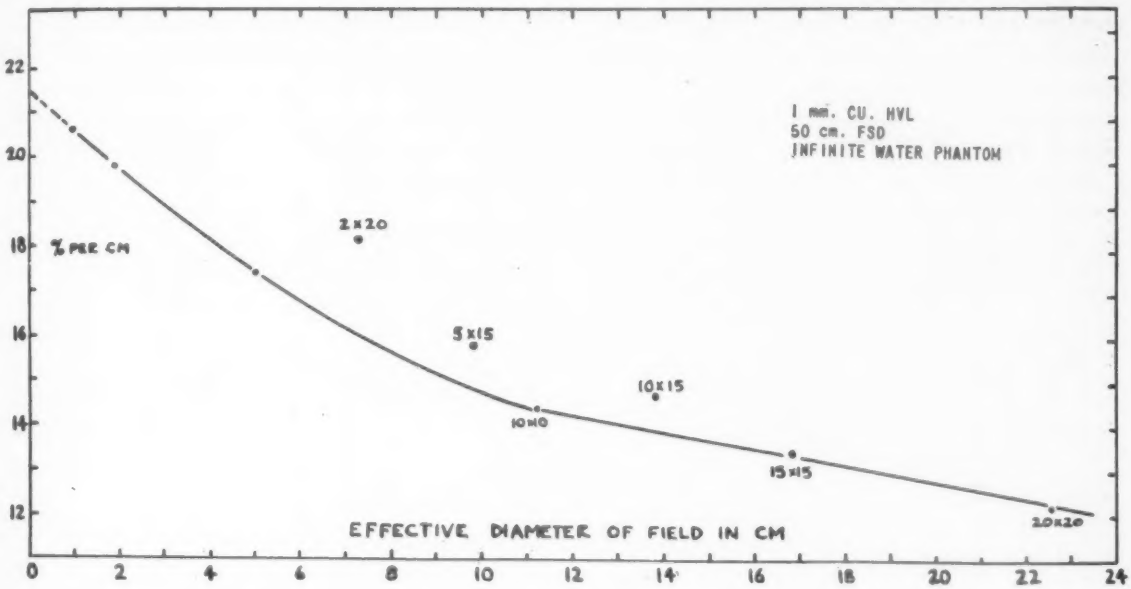

FIG. 6. Effective absorption coefficient as a function of field diameter. The three unlabeled points were circular fields.

as 100 per cent at the axis of the beam, decreases to about 90 per cent at 6 cm. from the central axis, and to almost 70 per cent at the edge of the field.

Whenever the attenuation curve is exponential, it can be characterized by an "effective absorption coefficient." Depth dose measurements have been made for several fields of different sizes, at the stated half-value layer and focus-skin distance, and the effective absorption coefficients are plotted in Figure 6. The curve connects the points for the square and circular fields. The smallest field measured was about 1 cm. in diameter. The curve may be extrapolated to zero field size, giving the absorption coefficient for the primary beam alone, an important physical characteristic of the beam. It is seen that, as might be expected, the effective absorption coefficient for the elongated fields corresponds to the absorption coefficient of a field smaller in area than the elongated field.

The assignment of a unique absorption coefficient to the primary beam deserves some explanation. It will be recalled that the attenuation of the primary beam is the

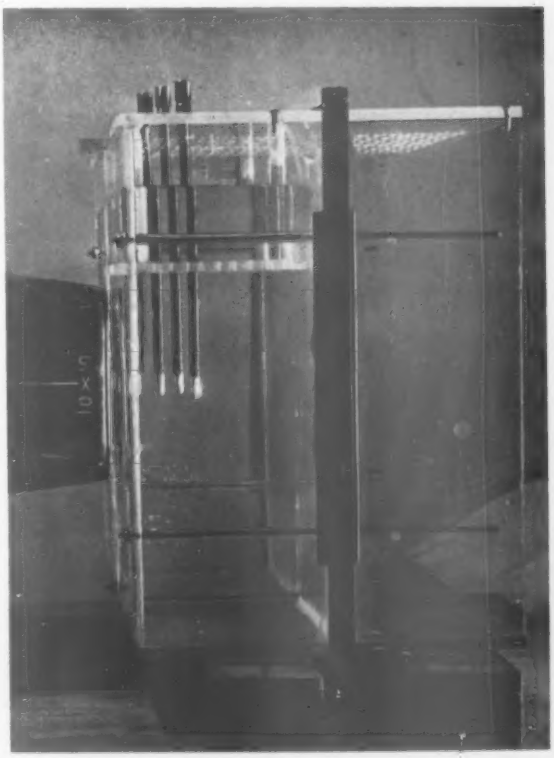

FIG. 7. The semi-infinite phantom, with three chambers and a treatment cone in place. The water level is about 3 cm. from the top. The depth in the direction of the beam is about 12 cm.

same as the attenuation of a very narrow, so-called "pencil" beam. Our smallest beam was about 1 cm. in diameter, and it showed exponential attenuation. It is assumed that a pencil beam would do likewise, since the extrapolation is short. Two factors tend to produce deviation from exponential absorption in the primary beam: The inverse square effect would make the attenuation *more* rapid, and a hardening of the heterogeneous beam would make the attenuation *less* rapid than exponential. The resultant of the two is apparently very close to exponential attenuation, at this half-value layer and focus-skin distance at least.

The measurement of depth doses in very narrow fields requires a precise alignment of field and beam axis. This is accomplished by use of fluorescent screen. Brass rods are substituted for the ionization chambers, one rod having a hole, and the other a point, at the level of the center of the ionization chambers. With the water level just below the beam center, it is relatively easy to check the alignment by viewing the screen on the distal side through the viewing window of the therapy room. When necessary, the beam axis and line of measurements were made to coincide within 1 millimeter.

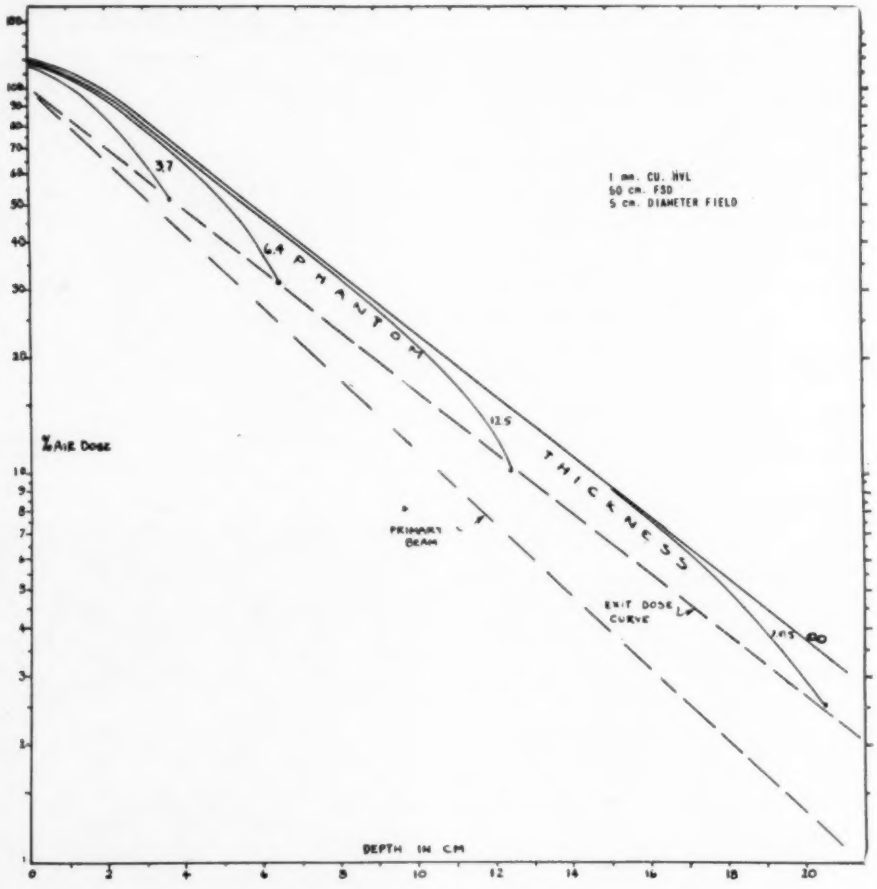

FIG. 8. Central axis depth dose curves in semi-infinite water phantoms of four different depths, using a 5 cm. diameter field.

THE SEMI-INFINITE PHANTOM

The first step in the transition from an infinite to a finite phantom is to reduce it to what we might call a plane, parallel, semi-infinite phantom. Such a phantom is shown in Figure 7. The term "semi-

infinite" is intended to describe the fact that the phantom is in effect infinite in all directions except in thickness. The phantom is made, and the chambers are supported, in essentially the same fashion as in the infinite phantom, except that the back is movable, may be set at any desired depth, and is sealed against the sides and bottom with paraffin.

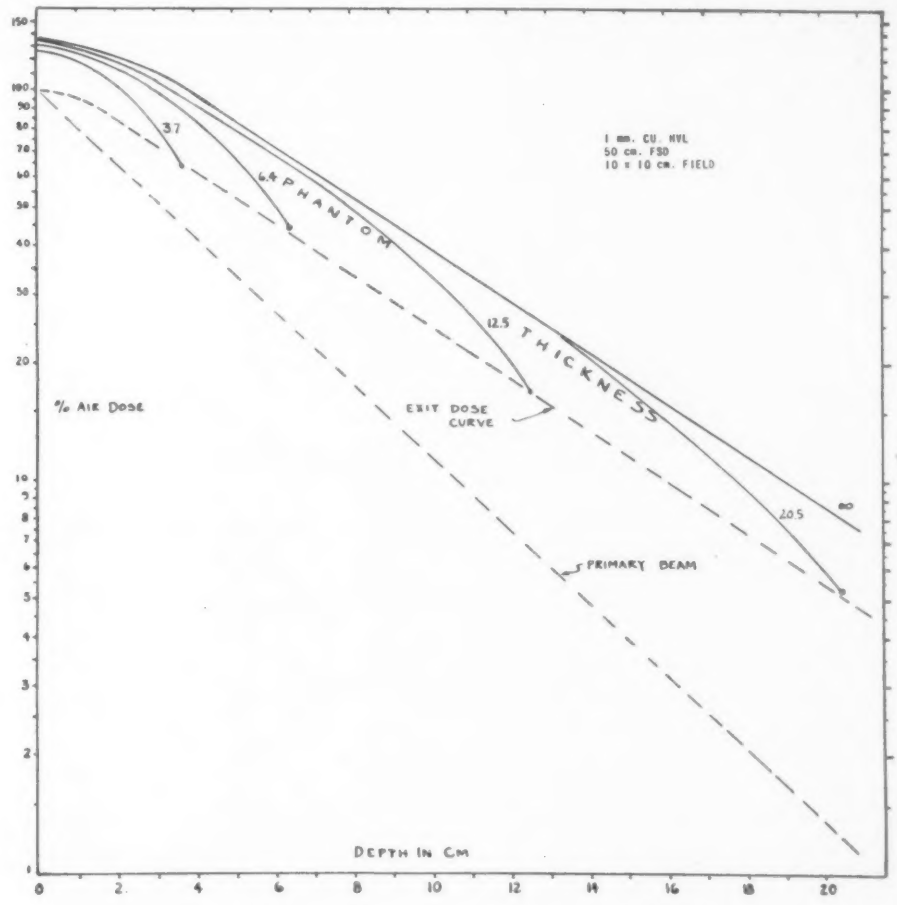

Fig. 9. Same as Figure 8, using a 10 by 10 cm. field.

In Figures 8, 9, and 10 are shown the results of measurements with this phantom. The percentage depth dose is plotted against the depth in centimeters. In contradistinction to the earlier graph, now backscatter has been included, that is, the air dose is taken as 100 per cent. The top curve is just the conventional, infinite phantom depth dose curve. The other solid curves are the experimentally determined central axis depth dose curves for the different phantom thicknesses. Such a curve, of course, terminates abruptly with the exit dose. The four observed exit doses are connected by a dotted line marked "exit dose curve." The dotted straight line, computed from the extrapolated absorption coefficient just described, represents the attenuation of the primary beam. Note that the exit dose curve is a straight line, accurately parallel to the infinite phantom depth dose curve, beyond 4 cm. The exit dose curve, like the primary beam curve, must go to 100 at zero depth.

There is considerable information in a graph of this type. For example, considering the 20 by 20 cm. field of Figure 10, the depth dose at 10 cm. may be considered to be made up of three components,

as follows: The primary beam alone at 10 cm. depth gives a dose of 12 r, for 100 r in air. Adding to that the forward scatter component brings the dose up to 34 r, which is the value of the exit dose curve at 10 cm. Then adding to that the saturated backscatter of an infinite phantom brings the dose up to 54 r, which is just the value of the infinite phantom depth dose curve at 10 cm. A finite phantom may have less than saturated backscatter, as, for example, with the 12.5 cm. thick phantom, where backscatter by the distal 2.5 cm. brings the total dose up to 49 r, which is just the value of the depth dose curve for the 12.5 cm. phantom at 10 cm. depth. Thus, in general, the difference between the exit dose curve and the primary beam curve, at a given depth, represents the dose due to forward scatter at that depth. The difference between any depth dose curve and the exit dose curve represents just the contribution due to backscatter. At the surface this difference takes on just the conventional backscatter value.

The fact that the infinite phantom depth dose curve, and the exit dose curve are parallel beyond about 4 cm. means that the backscatter contribution is a constant fraction of the total dose at these depths. At the surface, backscatter is a slightly smaller fraction of the total dose.

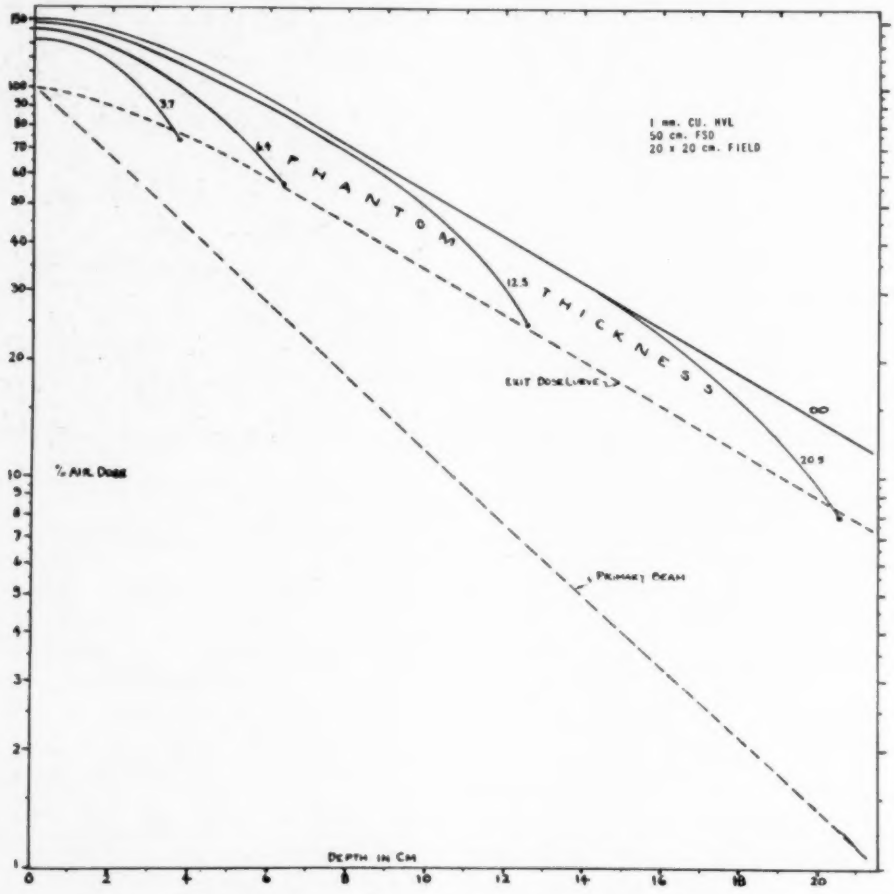

FIG. 10. Same as Figure 8, using a 20 by 20 cm. field.

The exit dose may of course be read directly from the graph for a phantom of any thickness.

Thus, with a graph of this type, the dose at any depth may be analyzed into its components for an infinite or a finite phantom. It is seen that for phantoms of 12 and 20 cm. thickness, the depth dose curve

has an exponential central portion, with a proximal transition region which lies below the exponential straight line due to unsaturated forward scatter, and a distal transition region which lies below the exponential line due to unsaturated backscatter. Since each of these transition regions is 4 to 5 cm. in thickness, the depth dose curve for the 4 and 6 cm. phantoms are lacking an exponential central portion.

From such a graph we can get an idea of how much deviation to expect from infinite phantom doses. The maximum deviation of course occurs for the exit dose, which for this field and half-value layer is always about 40 per cent less than the infinite phantom depth dose. It is interesting to note that for phantoms of 10 cm. or more in thickness, the central axis depth dose is within a few per cent of the conventional infinite phantom values, except for the last 4 cm. The smaller fields give qualitatively similar graphs, and the finite phantom measurements show relatively less deviation from the infinite phantom values. It was found that with these semi-infinite phantoms, too, the off-axis depth dose curves have the same shape as the central axis curves, and can be computed from the central axis curve simply by multiplying by a constant.

CIRCULAR PHANTOMS

The viewpoints and techniques illustrated in the discussion of infinite and semi-infinite phantoms, have been applied to phantoms of a more immediate clinical interest. In Figure 11 is shown a phantom representing the neck, used in studying dosage distribution in therapy of the larynx. The phantom is made of about 1/16 inch acrylic plastic, and is, of course, filled with water. A plastic tube, 16 mm. inner diameter, was placed in the approximate position of the trachea to study the effect of an air column.

Figure 12 shows the depth dose curve as measured in this neck phantom. (The dotted lines in the sketch show the lines along which the measurements were taken.) Note that the off-axis depth dose curves

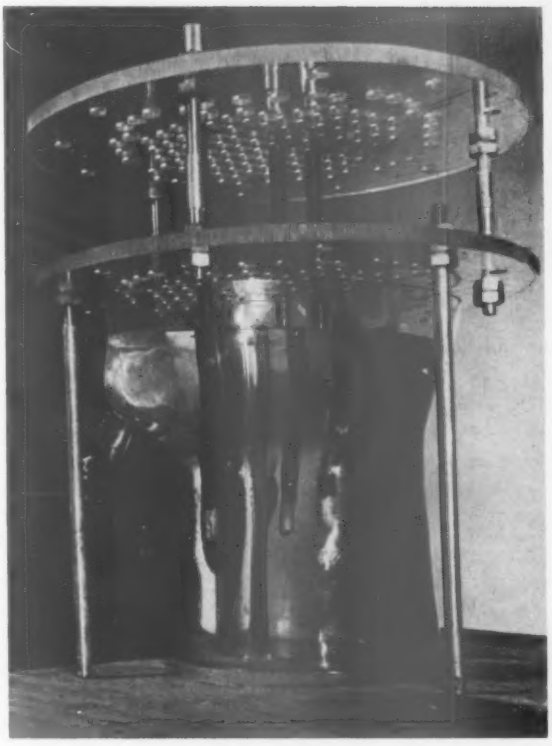

FIG. 11. Neck phantom with three chambers and treatment cone in place. The water level is about 2 cm. below the lower plastic sheet.

are parallel to the central axis curve, and that all these curves can be described in the same terms as were the curves in the finite, plane phantoms: there is an exponential central portion and a proximal and distal transition region. The dose posterior to the central axis is less than the central axis dose, as in an infinite phantom. However, the dose anterior to the central axis is greater than the central dose. The anterior tapering of the neck has the effect of shifting the depth dose curves without changing their shape. The line of measurement 1 cm. anterior to the central axis runs through the air column. The curve is displaced due to the absence of water, but distal to the air column the displaced curve resumes the slope and shape characteristic of all the other measured depth dose curves. The depth dose curve taken 4 cm. posterior to the central axis runs parallel to the others where it is inside the primary beam, but flops over where it crosses the edge of the diverging primary beam.

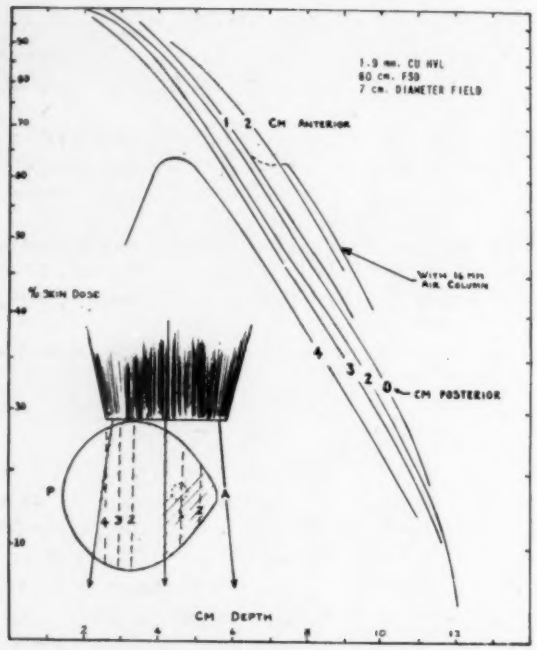

FIG. 12. Central axis and off-axis depth dose curves with the neck phantom. The sketch at the lower left is a schematic of the experimental set-up. The line of measurement of 4 cm. posterior to the beam axis crosses the edge of the primary beam, which accounts for the bend in the corresponding depth dose curve.

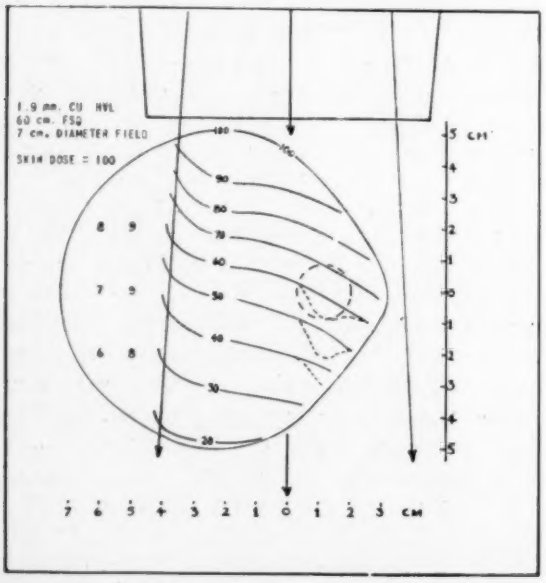

FIG. 13. Isodose curves for unilateral field in the neck phantom. The dotted lines show an air column and its effect on the isodose curves. The numbers outside the primary beam to the left indicate depth dose at those points.

Thus the practical significance of the present approach is that the task of securing the clinical dosage information in finite phantoms is not as formidable as is generally believed. It is only necessary to measure a central axis depth dose curve, and then take one set of measurements perpendicular to the central axis along the midline, to determine the entire dosage situation inside the primary beam, in this one plane.

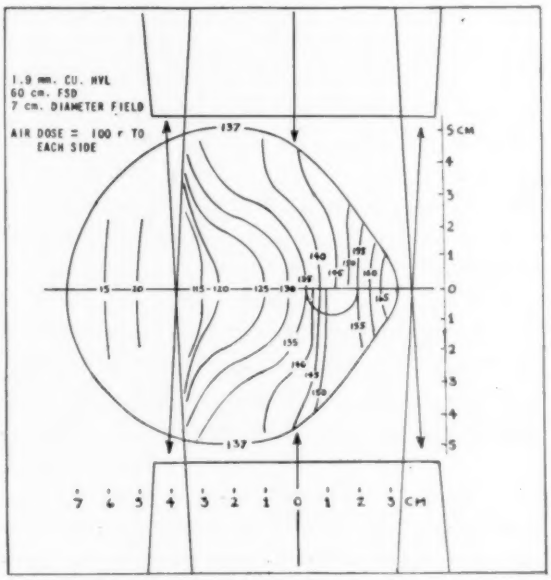

FIG. 14. Isodose curves for bilateral field in the neck phantom. The curves above the midline show the dose distribution in the normal phantom, and below the midline the curves show the dose distribution in the presence of the air column shown.

While all necessary dosage information is contained in the central axis and off-axis depth dose curves, for maximum clarity and usefulness it is necessary to translate this information into the familiar isodose curves. To do this is of course a matter of simple arithmetic only. Figure 13 shows such isodose curves for a single lateral field to the neck phantom. The dotted lines show the displacement of the isodose curves due to the air column. There was no measurable displacement except distal to the air column. Posteriorly, the isodose curves curl up at the edge of the beam,

in the usual fashion. Anteriorly, the isodose curves tend to follow the proximal contour of the phantom.

In Figure 14 are shown the isodose curves resulting from a bilateral application of this field. Above are shown the curves in the absence of the air column, and below are shown the isodose curves in the presence of the air column. Note that now the isodose curves have a general tendency to run parallel to the central axis of the beams. The air column has the effect of producing more uniform dosage anteriorly, and less uniform dosage posteriorly, without, however, changing the over-all decrease in dose in the anteroposterior direction, across the air column. The anterior taper of the neck of course produces a hot spot at the anterior surface on the midline, of about 170 r for 100 r in air to each of two lateral fields.

Measurements have also been made with cylindrical phantoms of 8, 10, and 12 cm. diameter, using on- and off-axis fields of various sizes. The results conform to the general viewpoint already given. The 10 cm. diameter cylinder was approximately the same size as the neck phantom, and gave results so nearly identical that it was clear that phantoms which are precise replicas of the body are not required. A simple geometrical shape should ordinarily be sufficient for clinical purposes.

CONCLUSION

Though it has not been possible thus far to compute theoretically a depth dose curve in an infinite phantom, an empirical approach has been developed over a period of years which is both convenient and adequate for clinical purposes. It is our belief that this empirical method can be generalized so that it will apply to finite phantoms in a fashion sufficiently simple to be useful to the clinician.

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DISCUSSION

Dr. William Harris, New York, N. Y. I suppose that to many of you this seems to be a step backward—that is, that anybody would have the temerity to still worry about 200 kv. with all the great enthusiasm about so-called supervoltages and radioactive isotopes, but there are still a few old-timers like myself who feel that voltages in the range of 200 kv. have still a great field of usefulness. It will probably be a long time before most of us will have apparatus in the range of a million or more, and also it is possible that the higher voltages may show no superiority over 200 kv.

This work by Drs. Wolf and Loevinger was stimulated by our desire to see whether we could approach homogeneous doses in a tumor. I suppose the same technique can be applied to any voltage in the future and we feel, from a practical point of view, that we have already learned something by this investigation; for instance, in the treatment of cancer of the larynx in which we are very much interested.

It is clear from these charts in the study of the larynx, that there is a "hot spot" in the thyroid cartilage with this technique and it probably explains the reason for the cartilage necrosis that one sees occasionally in the treatment of cancer of the larynx where there is no invasion by disease. It is the plan of Drs. Wolf and Loevinger, I believe, to apply this technique and study, to other parts of the body.

For instance, up to now, I imagine most people used charts on an empirical basis where most of the off-axis determinations were made by calculation. We plan to use this on extremities, in the pelvis, in the lungs, and in other parts where studies of this kind may point to a better technical approach to the treatment of cancer.

DEPARTMENT OF TECHNIQUE

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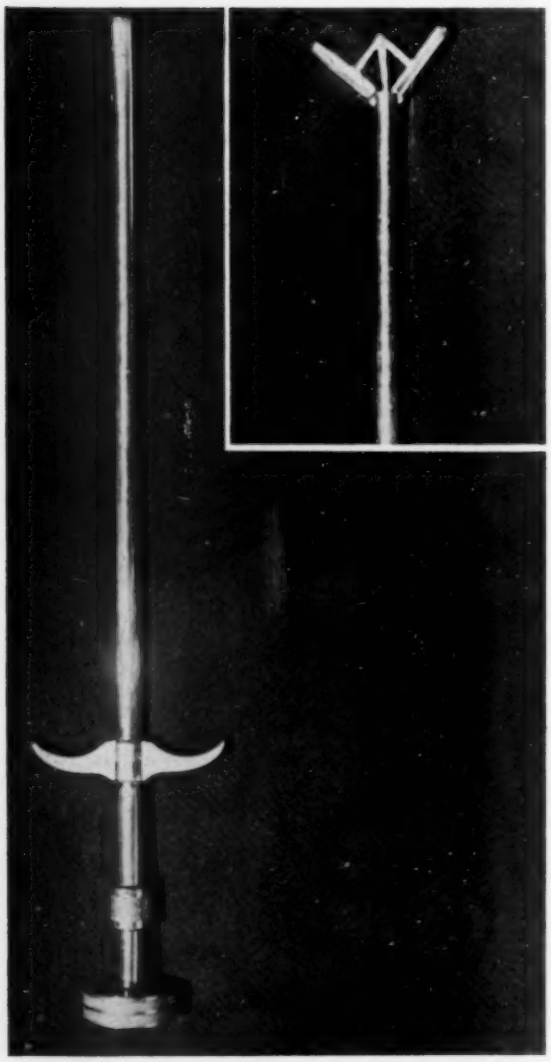
RADIUM NEEDLE THERAPY IN CANCER OF THE CERVIX UTERI

DESCRIPTION OF A NEW INSTRUMENT

By HOLLON W. FARR, M.D.,* *and* DOUGLAS J. ROBERTS, M.D.†
HARTFORD, CONNECTICUT



SINCE the report by Pitts and Waterman[1] on radium needle therapy in cancer of the cervix uteri, this has been the treatment of choice at this institution. External irradiation to pelvic fields is used as a preliminary measure to help in the control of node-bearing areas, particularly in the Stage II and III cases. To insure uniform therapy and results, most cases have been treated and supervised by the same group of men since 1936.

Our method has been to implant long parametrial needles lateral to the cervix and uterus and to implant shorter needles through the cervix into the uterus in a somewhat concentric manner, individualizing each case, thus attempting to administer a uniform dosage of radiation to the cervix, neck of the uterus and to each parametrial region. The needles used vary from 2 to 6 cm. in length, the number depending upon the ability to space these so as to avoid too great a concentration of dosage. A tandem intrauterine radium application is generally used in conjunction with the needles.

Needle insertion in the early case with mobile uterus can be surprisingly inexact

FIG. 1. The instrument is 12 inches long and has a diameter corresponding to that of a No. 6 Hagar dilator. The jaws open gradually to 180 degrees by turning the handle.

* Former Senior Assistant Resident in Surgery, Hartford Hospital.
† Radiologist, Hartford Hospital.

because of movement of the fundus when the cervix is anchored by the usual single-hook tenaculum. Likewise the technique in the frozen pelvis can be difficult for lack of visualization of the direction and depth of the uterine cavity. Absence of the cervix or replacement of the entire cervix with friable carcinoma offers another difficulty

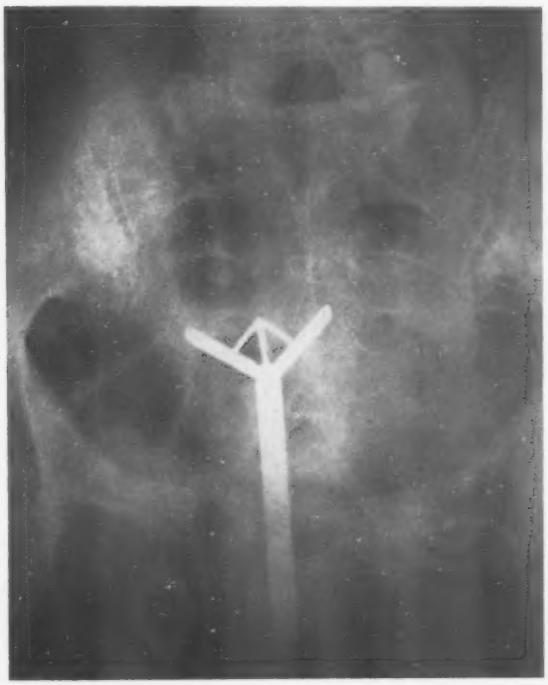


Fig. 2. Film showing tractor in position to start radium needle implantation.

in the proper placement of the needles. To correct these sources of error, one of us (H.W.F.) suggested the use of an old instrument for a new purpose.

The Young Straight Prostatic Tractor is a genitourinary instrument for the transurethral stabilization of the prostate in the perineal approach for prostatectomy, and is available at all large surgical instrument manufacturing concerns. It has proved to be of great benefit in our experience in completely stabilizing the uterus, as well as delineating the length and direction of the uterine canal.

The technique of using this instrument has been developed as follows: The cervix is grasped with a tenaculum, tissue for biopsy may be taken, the cervix is dilated,

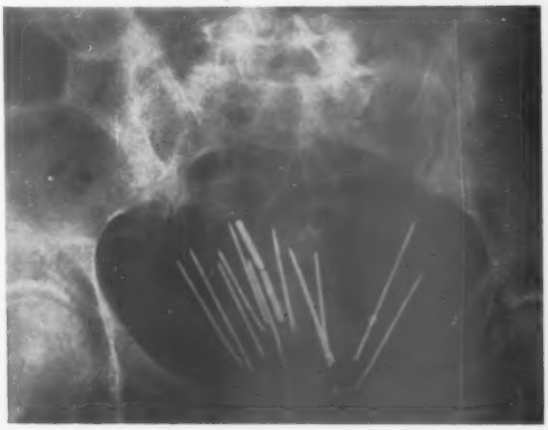


Fig. 3. Same patient showing radium needles in good position, a corona of twelve shorter cervical needle- and four parametrial long needles and tandem intrauterine applicator.

the tractor is inserted and the tenaculum removed, and the tractor jaws are spread. The last maneuver must be done gently, as there is danger of uterine perforation—an error which we have not experienced as yet. Parametrial and cervical needles are then inserted with relative ease and security without tenaculum interference. The instrument is then withdrawn and the radium tandem applicator inserted in the uterine canal enclosed in rubber tubing which is then stitched to the area of the external os. The fault of the instrument lies only in its efficiency, in that the operator has such tremendous leverage on the corpus that he tends to pull and thus prolapse it, distorting the normal parametrial structures as well as the fornices. Gentleness and precision will result in superior radium needle distribution.

It is conceivable that this instrument would also be useful in interstitial radiation with the newer radioactive substances in needle form, when they are available.

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Hartford, Conn.

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EDITORIAL

THE CAST SYNDROME

RECENTLY Dorph[1] reported a case of pernicious vomiting with fatal termination following application of a body cast and, in order to call attention to the possibility of such danger, he presented a full study of the problem referred to as the "cast syndrome."

The case of Dorph presented the following clinical course. A young girl, eighteen years of age, had been thrown from the handle bars of a motorcycle, sustaining a compound fracture of the lower third of the left femur. After two weeks' traction, she was placed in a body cast to immobilize the femur. Almost immediately she developed vomiting and three days later the cast had to be cut over the abdomen to relieve the pressure. The patient was discharged to her home in apparently good condition but had to be rehospitalized nearly two weeks later because of recurrent attacks of vomiting. An additional portion of the cast was cut out but after a short temporary relief she started to vomit every few hours. At this time a Levin tube was passed, leading to so much improvement that three days later the tube was removed and she was sent home. Within four days, however, she began to vomit again, at first periodically, and then almost continuously. She soon became comatose and on the twelfth day following her last discharge she was re-admitted to the hospital in an unconscious state. A diagnosis of acute alkalosis was made and emergency treatment instituted. The body cast had not been removed. Despite all measures taken the patient died six hours later.

The outstanding finding at the postmortem examination was acute dilatation of the stomach, associated with massive pulmonary edema and myocardial degeneration. The dilatation involved the entire stomach, which was the size of a small watermelon, as well as the duodenum up to the ligament of Treitz. Distally from this latter point the intestinal tract was normal in diameter. The transition from the greatly dilated caliber to the normal caliber was abrupt but there was no evidence of an organic obstructive lesion.

Acute dilatation of the stomach is known to occur secondary to numerous factors of a diverse character. Prior to the widespread employment of suction in the early postoperative period, it constituted one of the common and most serious complications in all types of surgical interventions. Thus Borchgrevink[2] in reporting in 1913 on a series of 138 collected cases found that in 66.6 per cent of them the acute gastric dilatation developed postoperatively, in 71 cases following laparotomies and in 21 cases following extra-abdominal operations. In the laparotomy group, operations on the female genital organs gave the largest and on the biliary system the next largest number, with 30 and 17 cases respectively, whereas in the extra-abdominal group there were 8 operations on the extremities, 7 on the thorax, 5 on the kidney and 1 on the face. Among the other causes listed by Borchgrevink were acute illness (15 cases), such as pneumonia, typhoid fever, etc.; chronic illness (5 cases), mainly tuberculosis and diabetes; deformity of the spine (7 cases); overloading of the stomach, usually in gluttonous

[1] Dorph, M. H. The cast syndrome; review of the literature and report of a case. *New England J. Med.*, Sept. 21, 1950, *243*, 440–442.

[2] Borchgrevink, O. J. Acute dilatation of the stomach and its treatment. *Surg., Gynec. & Obst.*, 1913, *26*, 662–693.

persons (11 cases); blow to the abdomen (2 cases); confinement (2 cases), and individual disposition without apparent cause (4 cases). A closer analysis of the patients with deformity of the spine shows that one had an enormous lumbodorsal scoliosis and 6 had spondylitic kyphosis with or without scoliosis. In the patient with lumbodorsal scoliosis there scarcely was space for a finger between the xiphoid process and the spine, and thus it was assumed that the deformity had produced a pressure on the stomach. In the other 6 patients, however, Borchgrevink was inclined to disregard the influence of the spinal disorder and he attributed the onset of the gastric dilatation to overloading of the stomach, since there was a history of excessive eating in nearly all cases. It is noteworthy that Borchgrevink, after such a thorough study of the problem, makes no mention of the possibility of an acute dilatation of the stomach arising from the effect of a body cast.

With the advent of early gastric intubation and efficient fluid-replacement therapy, postoperative acute dilatation of the stomach, to quote Wangensteen,[3] "has come to be a rare complication." Similarly, its incidence has greatly decreased in most of the other acute and chronic disorders enumerated above. There can be no doubt, however, that "the potential for its occurrence is ever present" and that continuous vigilance is necessary if one is to prevent a full development of the syndrome with its frequently fatal outcome.

Dorph,[1] in reviewing the literature on acute dilatation of the stomach due to the application of body cast, was able to collect 4 cases, 3 of which terminated fatally. The first case was described by Willett[4] in 1878. A young boy, seventeen years old, soon after being placed in a Sayre's body jacket to correct a kyphotic spine, developed pernicious vomiting and although the cast had been removed he died two days later. Postmortem examination revealed acute dilatation of the stomach without evidence of organic obstruction. In 1880 a similar observation was made by Buzzard[5] who applied a body cast to a twenty-two year old boy afflicted with Pott's disease. This patient felt relieved for a period of six days; then, after a copious meal, he suddenly developed severe vomiting and died in forty-eight hours, again despite the fact that the cast had been removed as soon as the vomiting started. Postmortem examination showed acute dilatation of the stomach and duodenum with a "kink" at the duodenojejunal junction which Buzzard interpreted as a pressure from the superior mesenteric artery. The third case was reported in 1901 by Kelling[6] in an article dealing with the mechanism of acute dilatation of the stomach. Kelling's patient, a girl sixteen years of age, had a full body cast applied for the correction of a congenital lumbodorsal scoliosis. Two days later she developed pernicious vomiting but after removal of the cast, withdrawal of 3 liters of fluid through a stomach tube and the administration of clysis, she started to improve and in two weeks had completely recovered.

The fourth case, described by Conley and Miller[7] in 1935, is unusual in the sense that the extreme dilatation of the stomach led to rupture of the organ with death resulting from peritonitis and septicemia. The case is that of a girl, fifteen years of age, who was admitted to the Cook County Hospital with a diagnosis of idiopathic scoliosis. She was placed on a Bradford frame with neck and pelvic traction and since there was no improvement on repeated roentgenographic examinations, at the end of ten weeks a body and neck cast was applied. After a few days she was dis-

[3] Wangensteen, O. H. Intestinal Obstructions. Second edition. Charles C Thomas, Springfield, Illinois, 1949, pp. 343–344.

[4] Willett, A. Fatal vomiting following application of plaster-of-Paris bandage in case of spinal curvature. *St. Bartholomew's Hosp. Rec.*, 1878, *14*, 333–335.

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[6] Kelling, G. Über den Mechanismus der akuten Magendilatation. *Arch. f. klin. Chir.*, 1901, *64*, 393–417.

[7] Conley, Arthur, and Miller, D. S. Acute dilatation of the stomach with perforation following body cast application. *Indust. Med.*, 1935, *4*, 185–186.

charged to her home in good condition. Nearly six weeks later, however, she had to be re-admitted as an emergency owing to a persistent vomiting associated with severe pain in the epigastrium. The cast was removed and following establishment of a diagnosis of acute dilatation of the stomach by means of a few swallows of barium mixture, Wangensteen intubation and fluid-replacement therapy was instituted. Parotitis and septicemia developed and the patient died eight days after re-admission. At necropsy multiple ruptures of the gastric wall were found with perforation of one of the ruptures into the abdominal cavity. The ruptures were located in the fundus which represented the weakest region of the greatly dilated stomach.

Dorph also cites the unreported case of a twenty-two year old woman observed at Temple University Hospital in Philadelphia who after being placed in a body cast for a lumbar injury developed a syndrome similar to that observed in his own case, likewise terminating in death.

Although it would seem to appear from Dorph's compilation that the so-called cast syndrome is comparatively rare it is probable that the condition occurs more frequently than is generally assumed. Conley and Miller,[7] for example, state that after observing their above described case they searched through the records of their hospital and came across 2 additional cases of acute dilatation of the stomach following application of the body cast. Wangensteen[3] feels that the potential for occurrence of acute dilatation of the stomach is so high that he routinely applies suction to an indwelling duodenal tube in all cases in which plaster of paris cast is applied following hyperextension of the spine.

Much has been written on the pathogenesis of acute dilatation of the stomach, especially as it concerns the postoperative form, but the problem still remains rather obscure and confused. Briefly, the following are the principal theories suggested: (1) Paralysis of the stomach. This is the oldest theory, first proposed by Brinton[8] in 1859 and expounded since then by many investigators. (2) Obstruction of the third portion of the duodenum by mesenteric compression. This theory was advanced by von Rokitansky[9] in 1861 who assumed that acute dilatation is simply a high obstruction caused by pressure of the mesentery and superior mesenteric vessels on the duodenum as it crosses the spine. (3) Reflex atony of the stomach with failure of resorption of the gastric and pancreatic juices. Dragstedt and his coworkers[10] presented evidence to the effect that the acute dilatation of the stomach is due to reflex inhibition of the peripheral gastric motor mechanism through efferent impulses reaching the stomach by way of the vagi and splanchnics. The atonic stomach fails to resorb the gastric and pancreatic juices and more particularly the sodium and chloride excreted in them. In certain cases a secondary mesenteric obstruction also occurs adding another obstacle to the passage of the juices into the lower bowel. (4) Air sucking due to relaxation of the superior esophageal sphincter. Morris, Ivy and Maddock[11] believe that the large quantities of air which are always present in acute dilatation of the stomach are best explained by assuming a relaxation of the superior esophageal sphincter while a negative intra-esophageal pressure is developed by inspiration. And there are still other theories.

It is probable that in the cast syndrome the acute dilatation of the stomach is the result of the hyperextended position of the spine and/or of the considerably reduced excursion of the abdominal muscles. By

[8] Brinton, W. Lectures on disease of the stomach. Churchill & Sons, London, 1859, pp. 291–300.

[9] von Rokitansky, C. Incarceration. In: Lehrbuch der pathologischen Anatomie. Third edition. W. Braumüller, Wien, 1861, vol. 3, pp. 187–191.

[10] Dragstedt, L. R. Acute dilatation of the stomach. In: Lewis' Practice of Surgery. W. F. Prior Co., Hagerstown, Md., vol. VI. Chapter 10, pp. 1–18; also, Dragstedt, L. R., Montgomery, M. L., Ellis, J. C., and Matthews, W. B. The pathogenesis of acute dilatation of the stomach. *Surg., Gynec. & Obst.*, 1931, *52*, 1075–1086.

[11] Morris, C. R., Ivy, A. C., and Maddock, W. G. Mechanism of acute abdominal distention; some observations. *Arch. Surg.*, 1947, *55*, 101–124.

following the theory of von Rokitansky both of these factors could lead to a compression of the third portion of the duodenum by the mesentery and the superior mesenteric vessels. A concomitant participation of the duodenum and stomach in the process of dilatation, as demonstrated at all postmortem examinations, lends considerable support to such assumption.

It is a surprising fact that roentgen examination has been used very little in the diagnosis of the cast syndrome. The only reference to a roentgen study is found in the article of Conley and Miller.[7] These authors administered a few swallows of a barium mixture and were able to visualize an enormously dilated stomach. They have also noted on the same roentgenogram, as an incidental finding, that the nasal tube used for suction was curled up in the esophagus, necessitating re-insertion into the stomach.

Although a clinical diagnosis of acute dilatation of the stomach is comparatively easy, especially if the condition occurs postoperatively or following overloading of the stomach, it appears nevertheless that roentgen examination has definite value under certain circumstances. In the cast syndrome, it aids in making a correct diagnosis at a stage which may mean the difference between life and death of the patient.

T. LEUCUTIA, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Pa. Annual meeting: Shoreham Hotel, Washington, D.C., Sept. 25–28, 1951.

American Radium Society
Secretary, Dr. J. E. Wirth, U. S. Marine Hospital, Baltimore, Md. Annual meeting: Hotel Claridge, Atlantic City, N. J., June 7–9, 1951.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y., Annual meeting: Palmer House, Chicago, Ill., Dec. 10–15, 1950.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6.

Section on Radiology, American Medical Association
Secretary, Dr. P. C. Hodges, 950 E. 59th St., Chicago, Ill.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Meets time and place Alabama State Medical Association.

Arizona Association of Pathologists and Radiologists
Secretary, Dr. R. Lee Foster, 507 Professional Bldg., Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Atlanta Radiological Society
Secretary, Dr. T. F. Leigh, Emory Univ. Hospital, Emory University, Ga. Meets monthly, except during three summer months, on second Friday evening.

Brooklyn Roentgen Ray Society
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, Oct. through April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. F. A. Riebel, 15 West Goodale St., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Columbus Athletic Club, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. B. D. Braun, 6 N. Michigan Ave., Chicago 2, Ill. Meets second Thursday of each month, October to April inclusive at the University Club.

Cincinnati Radiological Society
Secretary, Dr. E. C. Elsey, 927 Carew Tower, Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. J. R. Hannan, 10515 Carnegie Ave., Cleveland 6, Ohio. Meetings at 6:45 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Paul E. RePass, 306 Republic Bldg., Denver 2, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday Oct. and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. J. C. Cook, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. T. H. Lipscomb, 30 W. Beaver St., Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. R. C. Pendergrass, Americus, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Greater Miami Radiological Society
Secretary, Dr. David Kirsh, 712 duPont Bldg., Miami 32, Florida. Meets last Wednesday of each month at 8:00 P.M. at Veterans Administration Regional Office.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St. Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Kings County Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Wybren Hiemstra, 1414 S. Hope St., Los Angeles 15, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

* Secretaries of societies are requested to send timely information promptly to the Editor

LOUISVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

MAINE RADIOLOGICAL SOCIETY
Secretary, Dr. C. F. Miller, Central Maine General Hospital, Lewiston, Maine.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. G. A. Nicoll, 134 Apple St., Dayton, Ohio. Meets second Friday of fall and winter months.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary, Dr. J. W. Walton, Horton Memorial Hospital, Middletown, N. Y. Meets 8:30 P.M., fourth Tuesday each month, Sept. to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. T. J. Pfeffer, 839 N. Marshall St., Milwaukee 2, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, 572 Lowry Medical Arts Bldg., St. Paul 2, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. Downing, 816 Medical Arts Bldg., Omaha, Nebr. Meets fourth Thursday of each month at 6 P.M. at Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. L. L. Robbins, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Harvard Club.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. J. L. Olpp, 49 Ivy Lane, Tenafly, N. J. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

NORTH CAROLINA RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings by announcement.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB
Secretary, Dr. C. W. Wauters, 701 High St., Auburn, Calif. Meets at dinner last Monday, every second month, except June, July and August.

OHIO STATE RADIOLOGICAL SOCIETY
Secretary, Dr. E. C. Elsey, 927 Carew Tower, Cincinnati 2, Ohio.

OKLAHOMA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. W. E. Brown, 21st & Xanthus, Tulsa 4, Okla. Three regular meetings annually.

OREGON RADIOLOGICAL SOCIETY
Secretary, Dr. C. T. Jessell, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. G. P. Keefer, American Oncologic Hospital, Philadelphia 4, Pa. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary Dr. E. J. Euphrat, 3500 Fifth Ave., Pittsburgh 13, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

QUEENS ROENTGEN RAY SOCIETY
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, Sept. to May.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Fred Zaff, 135 Whitney Ave., New Haven, Conn. Meets bimonthly on second Wednesday.

RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY
Secretary, Dr. K. C. Corley, 1835 Eye St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, Jan., March, May, Oct. at 8:00 P.M.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. Peter Gianquinto, 685 High St., Newark, N. J. Meets annually at Atlantic City at time of State Medical Society and mid-winter at Elizabeth, N. J.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. A. V. Winchell, 40 Meigs St., Rochester, N. Y. Meets monthly on last Monday from September through May, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. H. R. Senturia, Pasteur Medical Building, St. Louis, Mo. Meets fourth Wednesday each month, except June, July, August, and September.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. S. H. Fisher, 107 E. North St., Greenville, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas. Meets Galveston, Jan 19 and 20, 1951.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

UTAH STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake

City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. R. C. Kiltz, 705 Medical-Dental Bldg., Everett, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

Wisconsin Radiological Society
Secretary, Dr. Irving I. Cowan, 425 E. Wisconsin Ave., Milwaukee 2, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-Ray Study Club of San Francisco
Secretary, Dr. M. A. Sisson, 450 Sutter St., San Francisco, 8, Calif. Meets Jan. to July, Lane Hall, Stanford Univ. Hospital; July to Dec. San Francisco Hospital.

Cuba, Mexico and Central America

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sanchez, Apartado No. 86 Panama, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. Jean Bouchard, Suite 305, 1555 Summerhill Avenue, Montreal 26, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. I. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. R. de G. Burnard, 170 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Asociacion Argentina de Radiologia
Secretary, Dr. Lidio G. Mocsa, Avda. Gral Paz 151, Córdoba, Argentina. Meetings held monthly.

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedad Argentina de Radiología, Filial del Litoral
President, Dr. Francisco P. Cifarelli. Meets monthly second Wednesday, 7:00 P.M., 663 Italia St., Rosario.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Colombiana de Radiologia
Secretary, Dr. Alberto Torres Focke, Av. 34, No. 17–34, Bogota, Colombia. Meets last Wednesday of each month.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Allies, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad. USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Societa Italiana di Radiologia Medica
Secretary Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

MID-WINTER RADIOLOGICAL CONFERENCE

The Third Annual Mid-Winter Radiological Conference, sponsored by the Los Angeles Radiological Society, will be held at the Biltmore Hotel, Los Angeles, California, on February 24 and 25, 1951. Non-radiological colleagues interested in any of the courses are, of course, invited to attend. The cost for the entire two-day session is $15.00. A banquet will be held on Saturday evening, February 25, at which the guest speaker will be Dr. W. Edward Chamberlain, Professor and Chairman of the Department of Radiology, Temple University, Philadelphia, Pennsylvania. A second out-of-state speaker at the Conference will be Dr. Edward B. D. Neuhauser, Roentgenologist, Infants and Children's Hospital, Boston, Massachusetts. The fee for the banquet will be $5.00. Reservations for the Conference and banquet should be accompanied by a check for $20.00 and sent to Dr. John B. Hamilton, 210 North Central Avenue, Glendale 3, California.

Hotel reservations may be made by applying directly to the convention manager of the Biltmore Hotel, Los Angeles, California. Early reservations are suggested since accommodations are limited.

INSERVICE TRAINING COURSE IN RADIOLOGICAL HEALTH

The School of Public Health of the University of Michigan is offering an Inservice Training Course in Radiological Health to be given on February 5–8, 1951.

This course is designed especially for the average health department personnel such as health officers, engineers and sanitarians, industrial hygiene personnel, and perhaps some nurses, with a view also to serving such workers in industry as find the indicated coverage sufficiently related to their problems. While the course is set up to meet a need presented by public health workers of Michigan, interested persons elsewhere are welcome.

Applications for enrollment in the course should be submitted in writing by letter addressed to H. E. Miller, School of Public Health, Ann Arbor, Michigan. The enrollment fee is $5.00. Applicants will be enrolled to the extent of seating space available. It is suggested therefore that persons planning to attend make early application.

Persons enrolling and completing the course will receive an official "Record of Attendance."

There are a limited number of rooms (most of them doubles) available at the Michigan Union. Persons wishing rooming accommodations should make application as early as possible direct to the University of Michigan Union, Ann Arbor, Michigan. In making application be sure to state that the accommodation is requested in connection with attendance of this course.

DR. L. R. SANTE HONORED

On September 26, 1950, Dr. L. R. Sante of St. Louis was honored by a reunion of radiological residents who had trained under him at Saint Louis University School of Medicine and the St. Louis City Hospital during the past thirty years. Eighty persons consisting of former residents and their wives assembled from all parts of the United States at the Sheraton Hotel in St. Louis to do honor to their former chief. Dr. William H. Stewart of New York, Dr. Sante's former teacher during World War I, acted as toastmaster for the occasion. Other speakers were Dr. Edgar Spinzig of St. Louis, Dr. Don English of Lima, Ohio, Dr. Arthur Echternacht of Fort Dodge, Iowa, and Dr. Don C. Weir, of St. Louis. During the evening Dr. and Mrs. Sante were presented with a beautiful television set.

ATLANTA RADIOLOGICAL SOCIETY

At a recent meeting of the Atlanta Radiological Society, the following officers were elected for the coming year: *President*, Dr. William W. Bryan, *Vice-President*, Dr. George Hrdlicka; *Secretary-Treasurer*, Dr. Ted F. Leigh.

INDIANA ROENTGEN SOCIETY

At the annual meeting of the Indiana Roentgen Society held on May 21, 1950, the following officers were elected for the year 1950–1951: *President*, Dr. Kenneth L. Olson, South Bend, Indiana; *Vice-President*, Dr. G. H. Wisener, Richmond, Indiana; *Secretary-Treasurer*, Dr. William M. Loehr, Indianapolis, Indiana.

AMERICAN RADIUM SOCIETY

The Thirty-third Annual Meeting of the American Radium Society will be held at the Hotel Claridge, Atlantic City, New Jersey, on June 7, 8 and 9, 1951.

CORRECTION

In the paper by Gilbert H. Fletcher entitled "The Planning of External Irradiation in Pelvic Cancer," published in the July, 1950, issue of the JOURNAL, pp. 95–111, Figure 17 on page 110 contains an error. The lower left hand column of Figure 17 should read as follows:

Fields narrowed to cm. wide
3 cm. from S 2000 r (air dose)
2400 r (skin dose)
Mx. T.D. 2400 × 1.4 = 3360 r
Mn. T.D. 2400 × 1.2 = 2880 r

ABSTRACTS OF RADIOLOGICAL LITERATURE

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INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

Head

GURDJIAN, E. S., WEBSTER, J. E., and LISNER, H. R.: Mechanism of skull fracture 1023
SCHOEPS, J.: Roentgen diagnosis of toxoplasmic defects 1024
OWEN, R. D.: Problem of hypopharyngeal carcinoma 1024
TOMPKINS, V. N., HAYMAKER, W., and CAMPBELL, E. H.: Metastatic pineal tumors 1025
ECKER, A.: Hyperostosing meningioma of pterion 1025
SMITH, H. V., and CROTHERS, B.: Subdural fluid as a consequence of pneumoencephalography 1025
PSENNER, L.: Clinical and roentgenological contribution on fetal adenoma of the hypophysis 1026
REYES, V., and HORRAX, G.: Metastatic melanoma of the brain 1026
POPPEN, J. L.: Cerebral arteriography 1026
CULBRETH, G. G., WALKER, A. E., and CURRY, R. W.: Cerebral angiography in "brain tumor suspects" 1027
GASS, H., *et al.*: Cerebral angiography recorded cinefluorographically 1027
FRANKEL, K.: Relation of migraine to cerebral aneurysm 1027

Neck and Chest

MALINER, M. M.: Table unit for fluoroscopic examination of infants 1028
LEWISON, E. F., and WEINBERG, T.: Carotid body tumors 1028
EISENBERG, R. B., and HORN, R. C.: Synovial sarcoma of the chest wall 1028
STEPHENSON, H. U., JR.: Malignant tumors of the parathyroid glands 1029
FULGHUM, C. B.: Cystic tumor of the thymic region and myasthenia gravis 1029
FRANKLIN, R. H., and TAYLOR, S.: Nonspecific granulomatous (regional) esophagitis 1029
BROMBART, M., GOUTKINE, J., and LAURENT, Y.: Gastric protrusions through the esophageal hiatus 1030
BERENBERG, W., and NEUHAUSER, E. B. D.: Cardio-esophageal relaxation (chalasia) as a cause of vomiting in infants 1030
MOTLEY, H. L., LANG, L. P., and GORDON, B.: Studies on the respiratory gas exchange in one hundred anthracite coal miners with pulmonary complaints 1031
DICKEY, L. B.: Pulmonary disease, associated with cystic fibrosis of the pancreas 1031
MOYER, J. H., and ACKERMAN, A. J.: Sarcoidosis 1032
THEODOS, P. A., *et al.*: Studies in the clinical evaluation of disability in anthracosilicosis 1032
FORSEE, J. H., MAHON, H. W., and JAMES, L. A.: Cavernous hemangioma of the lung 1033
HOCHBERG, L. A., and PERNIKOFF, M.: Primary chondromas of the lung 1033
BRAGG, E. A., JR., and LEVENE, G.: Hamartoma of the lung 1033
SHEINMEL, A., ROSWIT, B., and LAWRENCE, L. R.: Hodgkin's disease of the lung 1033
BONOFF, C. P.: Acute primary pulmonary blastomycosis 1034
STEIN, J., and SHEINMEL, A.: Cavitary disease of the lungs (due to less frequent etiological factors) 1034
COLLINS, MARTHA D., and FISHER, H.: Case of generalized hemangio-sarcomatosis erroneously considered as generalized tuberculosis 1034
BUCKLES, M. G., and NEPTUNE, W. B.: Tuberculous bronchitis in pulmonary resection 1034
HAMIL, B. M.: Bronchopulmonary mycosis 1035
SCHMIDT, H. W., CLAGETT, O. T., and McDONALD, J. R.: Bronchiolithiasis 1035
ERBER, B.: Endobronchial tuberculosis and the physiopathology of cavities 1035
MICHELIS, U.: Tuberculosis of the bronchial tree 1036
ARIEL, I. M., *et al.*: Primary carcinoma of the lung 1037
HEATON, T. G., and OGDEN, W. E.: Re-expansion of the artificial pneumothorax lung 1037
BREWER, L. A., DOLLEY, F. S., and EVANS, B. H.: Surgical management of chronic "spontaneous" pneumothorax 1037
OSWALD, N.: Pulmonary changes in the reticuloses 1038
LEVINSON, D. C., EDMEADES, D. T., and GRIFFITH, G. C.: Dissecting aneurysm of the aorta 1038

Zimmerman, H. A., Scott, R. W., and Becker, N. O.: Catheterization of the left side of the heart in man 1039
Heberer, G.: Disease process of pneumopericardium 1039
Wood, P.: Cardiac complications (discussion of the management of rheumatic fever and its early complications) 1040
Abbott, O. A.: Congenital aneurysm of superior vena cava 1040
Epstein, B. S.: Angiocardiography 1041
Dow, J. W., *et al.*: Studies of congenital heart disease. IV. Uncomplicated pulmonic stenosis 1041
Bogoch, A., and Christopherson, E. F.: Calcified cardiac aneurysms 1041

Abdomen

Kondo, B., *et al.*: Congenital coarctation of the abdominal aorta, a theoretically reversible type of cardiac disease 1041
Hodges, F. J.: Downward displacement of the gastric cardia 1042
Eibach, E.: Case report of polypoid gastritis .. 1042
LaDue, J. S., *et al.*: Symptomatology and diagnosis of gastric cancer 1042
Vogt, A.: Relationship of the frequency of gastric and duodenal ulcer during the past twelve years 1043
North, J. P., and Johnson, J. H., Jr.: Pyloric hypertrophy in the adult 1044
Shallow, T. A., Wagner, F. B., Jr., and Manges, W. B.: Primary carcinoma of infrapapillary portion of duodenum 1044
Pohlandt, K.: Detailed study of the juxtapyloric portion of the duodenum 1045
Mordeglia, M., Ferdman, J., and Entin, J.: Preferential localization in the duodenum of a case of lymphosarcomatous mycosis fungoides 1045
Prévôt, R.: Roentgen diagnosis of inflammatory lesions of the small intestine 1045
Dale, W. A., and Pearse, H. E.: Gas cysts of the intestines 1046
Buser, J. W., Kersner, J. B., and Palmer, W. L.: Carcinoma of the large bowel, analysis of clinical features of 478 cases, including eighty five year survivors 1046
Lawson, E. H., and Whitener, D. L.: Retrograde jejunogastric intussusception 1047
Denneen, E. V., and Broderick, T. C.: Gallstone intestinal obstruction 1047
Kay, S., and Talbert, P. C.: Adenoma of the liver, mixed type (hamartoma) 1047
Radakovich, M.: Epidermoid cyst of the spleen 1047

Gynecology and Obstetrics

Grünberger, V.: Clinical feature of multiple primary neoplasms in gynecology 1048
Tucker, Beatrice E., Solomkin, M., and Abrams, B.: Study of face presentation in the normal mature fetus 1048

ROENTGEN DIAGNOSIS

HEAD

Gurdjian, E. S., Webster, J. E., and Lissner, H. R. The mechanism of skull fracture. *J. Neurosurg.*, March, 1950, *7*, 106–114.

This study on the mechanism of skull fracture is based upon "stresscoat" studies describing the deformation pattern of the skull following impact. A more complete paper with the same title by the same authors is presented in *Radiology*, March, 1950, *54*, 313–339, which should be read in its entirety.

It was found that, "Whether a depressed fracture or a linear fracture is obtained depends entirely upon the velocity, the kinetic energy and, to a less extent, the shape of the injuring object. The area of impact is inbended, and in discrete regions of the skull there are evidences of outbending if the energy expenditure is adequate. If the area of inbending following impact rebounds without fracturing locally, a linear fracture may be initiated at a distance from the area of inbending due to tearing apart forces from outbending of the skull. The fracture then extends both toward the point of impact and in the opposite direction. If the area of inbending fails due to the magnitude and velocity of the blow, a depressed fracture is obtained. Linear fracture or fractures from outbending at a distance from the area of impact may be obtained if the velocity of the injuring object causing the depression is not too high. Tearing apart forces due to an impact are produced by bending of the bone and not by direct stress." —*Norman Heilbrun, M.D.*

Gurdjian, E. S., Webster, J. E., and Lissner, H. R. The mechanism of skull fracture. *Radiology*, March, 1950, *54*, 313–339.

Following impact to the skull the area of primary stress level is the weakest region and is the place where a fracture may be initiated. A single linear fracture is initiated away from the point of impact and extends toward the point of impact as well as in the opposite direction.

The area of secondary stress level is the region where a second fracture line may be initiated with additional energy. This is due to outbending peripheral to the point of contact. The areas of tertiary stress level are the regions where further fracture lines will be caused with expenditure of still more energy, resulting in a stellate pattern.

The authors describe the mechanism of linear, stellate, comminuted and depressed fractures. The patterns differ in different regions of same skull and occasionally in like regions of different skulls.

Clinical skull fractures are correlated with experimental fractures produced in cadaver skulls and dried skulls coated with "stresscoat" brittle lacquer.

Twenty-three figures of experimental and clinical skull fractures are included.—*E. E. Seedorf, M.D.*

Schoeps, Johannes. Die Röntgendiagnose der toxoplasmogenen Defekterkrankungen. (Roentgen diagnosis of toxoplasmic defects). *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, March, 1950, *72*, 577–586.

The term "toxoplasmic defects" includes all cases of physical or psychic defects ultimately attributable to toxoplasmosis and the ensuing defective healing. Because of the varied localization of the toxoplasmic encephalomyelitis or meningitis, many varieties of irritative neurological, degenerative psychic, or endocrinological conditions may arise. The roentgen diagnosis is based upon the typical course of the intrauterine hematogenous infection derived from the mother through the placental circulation, and having a marked predilection for the fetal brain and eye. Various regions of the brain, particularly the choroid plexuses, are involved, with the development of focal necrosis and eventual calcification. Hydrocephalus frequently results from the scarring of the healing process blocking the circulation of the cerebrospinal fluid.

Four cardinal points are involved in the diagnosis:

(1) Bilateral intracranial calcifications, particularly involving the choroid plexuses. These must be differentiated from calcification in the pineal, the falx, the hemangiomas of Sturge-Weber disease, arteriovenous aneurysms, arteries, brain tumors or cysts, tuberculomas, or calcified hematomas. Unilateral calcification in association with hydrocephalus is practically diagnostic. The calcifications may be demonstrated in newborn or premature infants.

(2) Hydrocephalus. Due to stenosis or obstruction of the aqueduct of Sylvius.

(3) Post-chorioretinitic eyeground changes (colobomata), other congenital degenerative ocular defects, including disturbances of innervation of the ocular muscles, and malformations such as microphthalmos.

(4) Antibody neutralizing and complement fixation tests. Positive findings do not persist for any great length of time and would not normally be found in an older child or adult.

Four cases are presented, the last of which is most interesting in that at the time of the original examination only one eye was involved with old chorioretinitis, but a few months later there was an acute febrile episode with acute chorioretinitis of the other eye, at which time positive serological tests were found, the only ones in the series. This attack was treated with a sulfonamide with some improvement followed by a relapse despite continuation of the therapy. Atabrine was then given and the chorioretinitis quickly subsided.—*J. J. B. Light, M.D.*

Owen, R. D. The problem of hypopharyngeal carcinoma. *Proc. Roy. Soc. Med.*, March, 1950, *43*, 157–170.

The author stresses the importance of thorough investigation of all patients presenting symptoms of "pricking sensations in the throat," voice changes of dysphagia. Although these symptoms may be produced by several other conditions the gravity of the malignancies of the postcricoid or hypopharyngeal region makes it essential that these lesions be ruled out by esophagoscopy.

The author presents 34 cases, all females, which he has followed for fifteen years. Their presenting symptoms were dysphagia and simple hypochromic anemia. All had direct examinations and dilatations to improve their swallowing; 23 are still alive with no evidence of malignancy; 5 have developed postcricoid carcinoma; 6 cases were lost as far as follow-ups were concerned with no evidence of carcinoma up to 1939. Patients with these symptoms must be followed closely for an indefinite period.

The preferred treatment for the malignant lesion is radical pharyngolaryngectomy. Recurrences are almost the rule in the simple pharyngectomy. Radiation therapy, he states, has been

of limited value in these cases. Two of the discussants of the paper believe that radiation therapy is beneficial and the results from this form of therapy are improving. The author's results were:

Pharyngolaryngectomy 22 cases 4–5 year survivals 18%
Lateral pharyngectomy 14 cases 2 year survivals 14%

The operative technique and plastic repair along with morbidity are discussed. Several good photographs of patients and of gross and microscopic specimen are included.—*F. M. Windrow, M.D.*

Tompkins, V. N., Haymaker, W., and Campbell, E. H. Metastatic pineal tumors; a clinicopathologic report of two cases. *J. Neurosurg.*, March, 1950, *7*, 159–169.

The authors report in detail, the clinical and pathological findings in 2 cases of pineal tumor in which metastasis to the lung had occurred. These were among 13 fatal cases of pineal tumors which reached the Army Institute of Pathology during World War II. Only 1 such case with metastasis had been previously reported in the literature.

In both cases the evidence indicated that the tumors were primarily intracranial and that the nodules in the lungs were metastatic. Although operative manipulation rather than natural mechanisms may have caused the introduction of tumor fragments into the venous blood stream, the nodules in the lungs were none the less metastases.

The question of the relationships of pinealoma to atypical teratomas and seminoma is discussed.—*Norman Heilbrun, M.D.*

Ecker, Arthur. Hyperostosing meningioma of pterion; clay model as aid in surgical excision without bone flap. *J. Neurosurg.*, March, 1950, *7*, 174–178.

The author used modeling clay in a prepared skull to simulate the hyperostosis of a meningiomatous plaque. This proved invaluable for orientation in both the preoperative study and during the actual course of the operation. In the case reported the hyperostosed bone of temporal fossa, anterior fossa, middle fossa and posterolateral aspect of the orbit was removed by direct excision without the use of a bone flap. The dural defect was closed by means of a pedicled graft made of pericranium and temporal fascia.—*Norman Heilbrun, M.D.*

Smith, H. V., and Crothers, B. Subdural fluid as a consequence of pneumoencephalography. *Pediatrics*, March, 1950, *5*, 375–389.

Pneumoencephalography may carry considerable risk which is usually justified, especially in neurosurgical work, by the information which it furnishes. However, in the study of neurological disorders of childhood, such as cerebral palsy, epilepsy and feeblemindedness, the risk must be evaluated before the examination is employed. The authors, after study of several hundred children with the above disorders, found the procedure well tolerated and the mortality negligible if no progressive disease was present and the hydrodynamics of the cerebrospinal fluid were normal.

However, they did find that in approximately one-third of their cases air entered the subdural space and that in some of these cases an air-fluid level appeared as the fluid was absorbed. In this latter group the children were found to take longer to recover from the diagnostic procedure and some became very ill. The authors discuss the diagnosis of the presence of subdural air and the differentiation of it from cerebral atrophy and hypoplasia. They emphasize that the bulk of the brain should be estimated from the size of the ventricles. They do not agree that the presence of subdural air indicates a pre-existing subdural hematoma unless the air is encysted or outlines a definite mass.

The mechanism of entrance of air into the subdural space is generally accepted to be due to technical error by which the bevel of the needle is partly covered by the arachnoid membrane which in turn acts as a valve allowing fluid to flow out through the needle but when air is injected it passes into the subdural space instead of the subarachnoid space. Rents in the arachnoid may be an alternate route for the entrance of subdural air.

Some mild reaction following encephalography due to mild meningeal irritation is the rule, but this is usually a short duration, subsiding in twelve to thirty-six hours. In the case of children in whom subdural air was noted the authors found that this reaction either did not subside or subsided and reappeared, persisting for well over a week. The children were pale and drowsy, irritable if aroused, and had projectile vomiting. These symptoms are not due

to the presence of the subdural air, according to the authors' experience. They studied the records of 75 children who had a total of 87 consecutive pneumoencephalographies. Subdural air was found in 37 percent of the cases. Over two-thirds of these cases were two years of age or under although the entire group was divided equally into those over and under two years of age. Eleven of the 31 cases in which subdural air was seen had disturbed postoperative courses. Several cases, in which a large quantity of subdural air was present had subsequent needling of the subdural space. Only 1 of 5 cases without symptoms had fluid in the subdural space. However fluid was obtained in every one of 6 cases needled in whom symptoms were present. This fluid was blood-stained and had a high protein content. The symptoms were not related to the degree of subdural air. The fluid found was indistinguishable from that found in subdural hematomas in children. The authors also felt that the symptoms exhibited by these children were compatible with such a diagnosis. Illustrative case reports are given.

The authors believe that the hematomas did not exist before the encephalography because of negative taps in several cases prior to encephalography; because the blood was fresh in the hematomas when removed; and because immediate benefit of symptoms was obtained following removal of the fluid. They suggest as the mechanism for the production of the hematomas that the cerebral veins passing from the brain to the superior longitudinal sinus are torn as the brain falls away from the cranium with the accumulation of air.

The authors feel that the danger of subdural hematomas is greater where the brain is atrophic.—*R. M. Harvey, M.D.*

PSENNER, L. Ein Beitrag zur Röntgenologie und Klinik des fötalen Hypophysenadenoms. (A clinical and roentgenological contribution on fetal adenoma of the hypophysis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, March, 1950, *72*, 586–590.

This is a case report, with abstracts of 5 additional cases from the literature. A rather large tumor had destroyed the sella and invaded the posterior portion of the sphenoid sinuses, producing the usual cerebral and ocular symptoms and amenorrhoea, with postoperative return of the menses.

These tumors may be quite small or very large, and are usually quite vascular. There are only minor endocrinological changes present, and those are of pituitary hypofunction due to pressure. The symptoms are frequently much less than would be expected from the size of the lesion. The tumors are benign and are not infiltrative in their growth. This is shown in the author's case in which the anterior portion of the sphenoid sinus was normal, although the posterior portion was occupied by tumor.—*J. J. B. Light, M.D.*

REYES, VICTOR, and HORRAX, GILBERT. Metastatic melanoma of the brain; report of a case with unusually long survival period following surgical removal. *Ann. Surg.*, Feb., 1950, *131*, 237–242.

The authors present the case of a fifty-seven year old man who in June, 1944, underwent surgical removal of a malignant black mole of the left shoulder region and left axillary adenectomy. The diagnosis was malignant melanoma involving the breast and axillary nodes. In February, 1945, the patient presented evidences of an intracranial space-taking lesion, but careful search of the remainder of the body failed to disclose any metastatic tumor. The patient was operated upon and a spherical, encapsulated, brownish-black tumor the size of a duck's egg was removed from the left frontal lobe. The patient made a good recovery with the exception of infrequent convulsive seizures which occurred about twice a year. When last seen, three years and nine months after the operation on the brain, the patient had no evidence of tumor and was carrying on his usual daily office work. The case is recorded because of the unusually long and useful survival period following the removal of a metastatic melanoma from the brain substance.—*C. L. Hinkel, M.D.*

POPPEN, JAMES L. Cerebral arteriography; editorial. *Surg., Gynec. & Obst.*, Feb., 1950, *90*, 248–250.

Cerebral arterial visualization was first described by Egas Moniz in 1927. The author has carried out the procedure in over 1,000 cases. Under pentothal anesthesia, 35 per cent diodrast is injected into the carotid artery. If the patient is sensitive to diodrast (as judged following injection of a small amount of the material into a vein or by application locally in the conjunctival sac), thorotrast is used.

The procedure is particularly helpful in diag-

nosing cerebral aneurysms (which have been bilateral in 2 per cent of the author's cases). Thrombosis of a major cerebral artery may be demonstrable by this method. Tumor and hemorrhage are indicated by displacement of vessels. In the case of hemorrhage larger vessels are displaced and there is an avascular region at the site of the hemorrhage.

Cerebral arteriography is not a substitute for air studies in determining the presence of a brain tumor but it is often a valuable adjuvant. It is of particular value in determining the relationship of the larger cerebral vessels to the tumor or whether a major vessel has been thrombosed as a result of the tumor.—*Mary Frances Vastine, M.D.*

Culbreth, George G., Walker, A. Earl, and Curry, Robert W. Cerebral angiography in "brain tumor suspects." *J. Neurosurg.*, March, 1950, *7*, 127–138.

The authors report a series of 96 angiograms in "brain tumor suspects" from the Division of Neurological Surgery at Johns Hopkins Hospital. In this procedure routine frontal and lateral stereoscopic films were made, necessitating four injections of contrast medium by the percutaneous puncture route. Films were made at one second intervals after the injection of 15 cc. of 35 per cent diodrast. Subsequent air studies or the course of the disease verified the angiographic diagnosis in all but five cases. The authors report excellent accuracy in correctly localizing the lesions which were present. There were 42 verified brain tumors, in all but three of which the angiograms permitted accurate localization. Tumors situated in the medial temporal lobes and in the midline structures are the least satisfactorily demonstrated by this technique.

In the majority of cases it was not necessary to do air studies as the arteriograms proved adequate for localization and diagnosis of the tumor. Arteriography causes less discomfort to the patient than air studies, indicates the position of the major arteries to the neurosurgeon and is of particular value in vascular meningiomas, certain of the gliomas and arteriovenous angiomas.

The paper is excellently illustrated, and the angiographic characteristics of certain tumors are clearly presented.—*C. L. Hinkel, M.D.*

Gass, H. Weinberg, S., Craig, A., Thompson, J. J., and Dreisinger, F. Cerebral angiography recorded cinefluorographically. *J. Neurosurg.*, March, 1950, *7*, 139–145.

The authors report the development of a successful cinefluorographic method of cerebral arteriography. They have used this method in 20 cases and obtained angiograms which they consider satisfactory for interpretation and clinical use. The cinefluorographic unit consists of a 35 mm. camera with a f/0.85 lens and a Dupont-Patterson E-2 screen. The camera speed is 60 frames per second which produces moderate slow motion when projected. The injection is carried out through the percutaneous route, only one injection being used as a general procedure. A continuous exposure of five seconds is utilized which is long enough to visualize the entire arterial phase. The authors prefer 50 per cent diodrast. By this method it is possible to see on a continuous record, the filling of the internal carotid and the significant arterial branches within the cranium.

The image produced lacks the sharpness and definition of good still angiograms but, in the opinion of the authors, the legibility is adequate for clinical purposes. The lack of steroscopic views is compensated for by purposeful rotation of the head during the exposure whenever indicated. Anteroposterior views have not been attempted thus far.

It is felt that the advantages of this method over the conventional methods are that only a single injection must be used and the radiologist obtains a continuous record of filling of the arterial tracts. Obviously the employment of such a technique is based upon the availability of a specially constructed cinefluorographic apparatus, such as is in use at the Strong Memorial Hospital, Rochester, N. Y.—*C. L. Hinkel, M.D.*

Frankel, Kalman. Relation of migraine to cerebral aneurysm. *Arch. Neurol. & Psychiat.*, Feb., 1950, *63*, 195–204.

Three cases of recurrent headache are reported in which intracranial aneurysm was present. The author discusses the relation of headache to intracranial aneurysm and indicates that a presumptive diagnosis of intracranial aneurysm can be made clinically before the appearance of the usual signs of rupture. In reviewing these cases a history of three distinct types of headache was found. Initially there is episodic headache, of years' duration. This is followed by a change in the type of the head-

ache one to several months before the patient seeks hospitalization, the change being increased severity, frequency or duration of the headache and at times associated with a change in location. Finally, there appears the severest and most persistent headache often accompanied with paralysis of extraocular muscles.

In all these cases the nature of the headache, which had been present for periods of from twelve to fifty years, changed from one to several months before the patient entered the hospital. This change should arouse suspicion of the development of an intracranial complication, to be interpreted as permanent dilatation of a congenitally weak portion of the arterial wall or dilatation of a minute aneurysm. It is suggested that the patient be closely observed and possibly subjected to diagnostic procedures at that time. There is a further change in the character of the headache at the time of massive dilatation and/or rupture of the aneurysm usually accompanied with palsy of the extraocular muscles, when the diagnosis of the intracranial aneurysm is made more readily—*K. K. Latteier, M.D.*

NECK AND CHEST

Maliner, M. M. A table unit for fluoroscopic examination of infants. *J. Pediat.*, Feb., 1950, *36*, 228–229.

This article describes a table unit devised by the author for fluoroscopic examination of the chest of infants in the erect position. The apparatus consists of a table upon which is mounted a removable rotating seat with sliding arm supports. The latter can be adjusted with thumb screws snugly against the side of the patient, thus restricting arm motion and providing immobility in the erect position. The seat can be rotated so as to provide immobilization of the infant in the various oblique positions for cardiac fluoroscopy.

Anyone interested in a support which permits fluoroscopy of the infant in the erect position is referred to the original article for details of the construction of the apparatus which is adequately illustrated.—*R. M. Harvey, M.D.*

Lewison, E. F., and Weinberg, T. Carotid body tumors; a case report of bilateral carotid body tumors with an unusual family incidence. *Surgery*, March, 1950, *27*, 437–448.

Carotid body tumors are thoroughly discussed and the topics included are: history, anatomy, histology, embryology, pathology, physiology, etiology, diagnosis and treatment. A case is reported that had bilateral carotid body tumors. Three of the patient's first cousins on his father's side had proved unilateral carotid body tumors.

The carotid body, which is bilateral, is located at or near the bifurcation of the common carotid artery and is closely associated with the great vessels of the neck and the vagus, hypoglossal and glossopharyngeal and other major nerves. It is the largest of the paraganglia included in the chromaffin system. Aberrant carotid bodies may occur anywhere along the sympatheticochromaffin system. The carotid body is thought to arise from the neural tube as primitive cells of the sympathetic system and differentiate into the chromaffin and sympathetic ganglia cell masses. It is thought to be a sensory organ containing chemoceptors which are sensitive to changes in oxygen tension of the blood and through which reflex changes in respiration are effected. However, no physiological respiratory or circulatory change was noted when bilateral carotid body tumors were removed. The function of the carotid sinus is entirely separate and different from that of the carotid body.

Excepting tumors, no other disease of the carotid body is known. The tumors are mostly benign and contain few mitotic cells. They are slow growing and usually envelop the adjacent vessels and nerves. If not completely removed they tend to recur.

The clinical diagnosis is seldom made and then with little degree of certainty. The possibility of a carotid body tumor should be kept in mind in all tumefactions of the upper anterior cervical triangle that are partially covered by the sternocleidomastoid muscle.

The treatment is usually surgical and the mortality is very high. Early postoperative death is as high as 30 per cent and more than 80 per cent of the patients who survive the operation are left with some marked postoperative disability such as aphasia or hemiplegia. Bevan and McCarthy reported a case diagnosed at operation as a carotid body tumor which was not excised and which received radiation therapy with an apparent cure. Others report no startling results from roentgen and radium therapy.—*C. W. Cooley, M.D.*

Eisenberg, R. B., and Horn, R. C. Synovial sarcoma of the chest wall. *Ann. Surg.*, Feb., 1950, *131*, 281–286.

A case of synovial sarcoma arising in the chest wall of a six year old white female is reported. Synovial sarcomas have been observed in the extremities, axillae, groins, and buttocks. The condition is rare and is of interest to the radiologist in the differential diagnosis of soft tissue tumors. The pathologic picture of the tumor and of the recurrences is clearly described. Synovial sarcomas may arise at sites far removed from regularly occurring synovium-lined structures and it is theorized that such tumors may be a neoplastic expression of the ability of the supporting tissues of the body to form synovium-lined spaces under certain conditions.—*G. P. Keefer, M.D.*

Stephenson, Hack U., Jr. Malignant tumors of the parathyroid glands; review of the literature with report of a case. *Arch. Surg.*, Feb., 1950, *60*, 247–266.

The author has reviewed the literature because of the lack of definite criteria as to what constitutes parathyroid carcinoma. Nineteen cases were found which were considered valid. These were divided into 9 non-functioning and 10 functioning tumors. To the latter group he has added an additional case.

It is interesting that in the non-functioning tumors a cervical mass had been present and noticed by the patient for periods of from two months to thirty-five years and had been the reason for medical consultation. In contrast, the patients with functioning tumors sought medical attention for reasons related to hyperparathyroidism. With hyperfunction the tumor had rarely been noticed by the patient and was not always recognized on physical examination. The non-functioning tumors were usually the large ones. The tumor reappeared in the neck after operation, usually within a year, in all but 5 instances.

The incidence of malignant tumor is distinctly rare as compared to adenoma. The evidence of the malignant character of a parathyroid tumor lies not so much in the cellular picture, but rather in evidence of invasion by the tumor cells of the capsule or the lymphatic or blood channels. Because of this it is necessary for the tumor to be completely removed surgically. This is especially true when the tumor is adherent and should imply removal of the adjoining thyroid. Remote metastasis occurs most often to the lungs and is usually late as it is the nature of the tumor to manifest its malignant character first by invasion of contiguous structures.—*Norman Heilbrun, M.D.*

Fulghum, Charles B. Cystic tumor of the thymic region and myasthenia gravis; report of a case in which the tumor was removed. *Ann. Int. Med.*, Feb., 1950, *32*, 327–335.

The author describes a case of congenital cystic tumor of the thymic region which is not definitely of thymic origin. According to the pathologist's report a careful study failed to reveal any evidence of Hassall's corpuscles or other structures identifying the tumor as being with certainty of thymic origin. This fact only serves further to confuse the existing facts concerning the relief of symptoms of myasthenia gravis by thymectomy.

The present knowledge can be summarized by stating: (1) that surgical removal of a tumor of the thymic region does seem to influence the course of associated myasthenia gravis and that the best chances of recovery are to be expected in patients who have not had the disease for an extended period (i.e., not over one year), and (2) that those patients from whom thymic tumors are removed do no better than those without tumors from whom the entire gland is removed.—*H. R. Fishback, Jr., M.D.*

Franklin, R. H., and Taylor, S. Nonspecific granulomatous (regional) esophagitis. *J. Thoracic Surg.*, Feb., 1950, *19*, 292–297.

The intrathoracic causes of dysphagia in the adult usually fall into one of three main groups: cardiospasm, neoplasm, and stricture following ingestion of a corrosive. Recently interest has been shown in spasm or stricture associated with peptic ulceration of the esophagus.

The 3 patients described fall into a different category, and they all fail to show any association with tuberculosis, syphilis, or peptic ulceration; nor had any of them swallowed a corrosive. A feature common to these 3 cases is production of a nonspecific granulomatous lesion in the esophagus.

In the first case discussed, the clinical, roentgenologic, and esophagoscopic appearances all suggested a carcinoma of the esophagus. If biopsy had been omitted and radiotherapy instituted, a cure by this treatment would have been claimed. The roentgenologic findings in the other 2 cases were strongly suggestive of neoplasm, even though both patients were young (aged thirteen and thirty-three years, respectively).

The cause of this lesion is obscure. In 2 patients the onset of dysphagia was preceded by a period of mental stress. Geographically they were widely distributed: England, Sweden, and India. However, the histopathologic picture was similar in all three. One is tempted to compare it with regional ileitis.

The importance of this observation is its clinical and roentgenologic mimicry of cancer. The diagnosis of esophageal carcinoma cannot be made without a histopathologic examination.—*S. N. Tager, M.D.*

BROMBART, M., GOUTKINE, J., and LAURENT, Y. Les protrusions gastriques à travers l'hiatus oesophagien. (Gastric protrusions through the esophageal hiatus.) *Acta gastroenterol. belg.*, March, 1950, *13*, 222–244.

After a brief review of the anatomical features favoring the formation of hiatal hernias, the authors discuss the accepted classification into three types: (1) short esophagus with part of the stomach above the diaphragm; (2) paraesophageal herniation along either side of a normal esophagus; and (3) gastric herniation due to an elevated esophagus of normal length.

To these should be added the intermittent reducible small gastric protrusions due to mild insufficiency of the hiatus, which Guttmann calls "engagements à éclipse," and which are thought by Åkerlund to represent a simple physiological condition due to age and increase of intra-abdominal pressure.

The symptoms may be absent, very mild, or marked. The pain is usually retrosternal or epigastric with possible radiation to the left arm.

Gastric hernia can be associated with any pathological condition of the gastrointestinal tract, such as diverticulosis, cholelithiasis, gastric ulcer, anemia, or angina pectoris.

All conventional procedures useful in searching for an hiatal insufficiency are reviewed with special stress on swallowing barium in the ventral Trendelenburg position.

Differential diagnosis must be made from epiphrenic diverticulum and from the so-called "phrenic ampulla," the latter, as masterfully described by Templeton, representing a physiological dilatation which occurs under definite circumstances of artificial "blockage" created, for instance, by the Valsalva test.—*Louis-Philippe Bélisle, M.D.*

BERENBERG, W., and NEUHAUSER, E. B. D. Cardio-esophageal relaxation (chalasia) as a cause of vomiting in infants. *Pediatrics*, March, 1950, *5*, 414–420.

Roentgenologic study is warranted in infants who vomit repeatedly without satisfactory explanation. The authors have encountered 24 patients in a five year period with repeated vomiting, apparently due to persistent relaxation of the hiatal portion of the esophagus. They use the term chalasia for this condition. The mechanism producing the relaxation is considered in detail and the authors conclude that in the majority of cases the basic fault is temporary neuromuscular dysfunction.

Clinically the syndrome is characterized by the onset of vomiting within three to ten days after birth. The vomiting is progressively worse although the nutrition of the infant does not suffer. The vomiting is effortless and rarely projectile. The vomiting is more apt to occur when the infant is in the supine or decubitus position. All other studies to find a possible source for the vomiting were negative. Feeding changes were valueless. The most important diagnostic aid is the fluoroscopic examination of the swallowing function of the esophagus. Barium is given in the formula or in 5 per cent glucose through the nursing bottle. The esophagus is dilated and peristaltic waves are reduced in intensity and frequency. No pinch-cock action of the cardio-esophageal sphincter is seen. With inspiration barium is forced back into the esophagus through the widely patent cardia. With the patient recumbent or when pressure is made over the abdomen the same regurgitation occurs. Regurgitation does not occur in the erect position.

The condition can be distinguished from congenital shortening of the esophagus by the absence of gastric mucosa above the diaphragm. Persistent relaxation of the hiatus may occur in the presence of obstructive lesions of the pylorus or duodenum, but these should be easily recognizable. The chief complications encountered were dehydration, failure to gain weight, and mild acidosis. Hematemesis did not occur and there were no fatalities. Drugs of the atropine and prostigmine groups were ineffective.

The treatment consists in feeding the child in the sitting or semi-erect positions and maintaining this position for at least thirty minutes after the feeding is complete. Thickening of the formula is also of value. All of the patients in this group responded to this regimen and were usually cured after three to four weeks. Subsequent roentgen studies showed a return of the

normal hiatus tone in most cases.—*R. M. Harvey, M.D.*

Motley, H. L., Lang, L. P., and Gordon, B. Studies on the respiratory gas exchange in one hundred anthracite coal miners with pulmonary complaints. *Am. Rev. Tuberc.*, Feb., 1950, *61*, 201–225.

Studies were made on a series of 100 anthracite coal miners, all with a long history of work in the mines. These were unselected except that cases with proved tuberculosis, malignancy or other disabling conditions were omitted.

On studies of expired air and arterial blood an attempt was made to determine the degree of impairment of respiratory gas exchange between alveoli and arterial blood, and to correlate this information with the degree of pulmonary emphysema. The diagnosis of emphysema is based on the relationship of the volume of residual air to vital capacity and total capacity, in accordance with a previously published article by these authors. Six representative cases are presented in detail with roentgenograms.

These studies on respiratory gas exchange revealed a lack of correlation between the extent of function impairment and the roentgenographic findings (inspiration and expiration films), and also between the extent of function impairment and the degree of pulmonary emphysema.

In approximately one-third of the cases studied, the disability arose from fibrosis and the accompanying unequal alveolar aeration and perfusion (the distribution factor), which decreased the amount of oxygen in the arterial blood. This factor was important regardless of the degree of emphysema. Arterial oxygen saturation was markedly decreased after exercise in some cases in which it was normal during rest.

Polycythemia and elevation of hemoglobin level were rare findings even in the presence of advanced emphysema, reduced ventilatory capacity, arterial oxygen unsaturation, and marked dyspnea on exertion.

No single test is adequate for evaluation of the degree of pulmonary function impairment. Measurements of ventilation, residual air, oxygen uptake from the air breathed, and arterial blood studies both during rest and exercise are required. Occasionally measurements on pulmonary circulation will be needed.—*Lois C. Collins, M.D.*

Dickey, Lloyd B. Pulmonary disease, associated with cystic fibrosis of the pancreas. *Dis. of Chest*, Feb., 1950, *17*, 151–171.

Knowledge of cystic fibrosis of the pancreas and its associated lesions as an entity is fairly recent. It is much more common than formerly thought to be, and was found in about 3 per cent of the necropsies at the Babies Hospital in New York. In 60 per cent of these the respiratory symptoms and pulmonary disease had masked the pancreatic disease.

The typical symptoms are chiefly those of early failure to gain on an adequate diet, with unimpaired appetite, with steatorrhea, soon followed by symptoms of chronic respiratory infection. The latter are the symptoms for which the patient is often brought first to the physician, and they very soon dominate the clinical picture. The cough resembles that of pertussis and this disease is often suspected. The thick tenacious mucus is similar in consistency to that found in whooping cough.

An examination of the duodenal contents always shows absent or greatly diminished trypsin activity, and is always necessary to make certain the diagnosis. This examination should be repeated at several intervals for substantiation.

The roentgenological findings are extensive. Barium in the intestinal tract may reveal complete obstruction during the newborn period if the mucus in the meconium is viscid enough to cause the condition known as meconium ileus. This condition is fatal and is present before pulmonary disease is manifest. If the condition is less severe, the typical picture of vitamin deficiency may show in the intestinal tract as "laking," with air and fluid levels and markedly slow bowel motility. Loops are dilated, and the distal small bowel is obviously smaller than the distended portions. Neuhauser has described the extensive pulmonary changes and divides them into two stages. In the first there is evidence of bronchial plugging without much infection. There is flattening of the diaphragm and many portions of the lung will appear emphysematous. There may be bulging of a few or many of the intercostal spaces. The lungs remain hyperexpanded in expiration, and the picture of obstructive emphysema with poor air exchange may be recognized. Areas of lobular atelectasis may be present, and lobar collapse is frequent. The second stage shows infection supervening, with increased hilar shadows and prominence of the bronchovascular markings.

Thickened bronchial walls may occasionally be seen and areas of peribronchial increased density. Although bronchiectasis is usually present, Neuhauser does not believe the use of lipiodol is warranted to confirm this.

The pathological conditions found in cases coming to necropsy are extensive. There is always a fibrotic or cystic pancreas in which the ducts may be open or in a state of atresia. Either small multiple abscesses, bronchiectasis often more marked in the upper lobes, or chronic pneumonia, or combinations of these are present. The primary feature is the production of abnormal secretion, with the morphological changes secondary. They believe the pulmonary disease primarily due to dietary factors, partially but not purely to lack of vitamin A.

The treatment of this disease should be early and vigorous. The diet should be dictated by the pancreatic deficiency, and lies within the province of the pediatrician or the general practitioner. Large amounts of vitamin A should be given by mouth in a water soluble form, and intramuscular therapy with vitamins is not necessary except possibly initially. If pulmonary symptoms are marked when the patient is first seen, the situation may be a matter of immediate survival. It is often necessary to administer oxygen, and to remove the tenacious bronchial secretions by mechanical suction. Both the immediate and long-time treatment of the pulmonary complications should consist of the giving of penicillin.—*E. J. McDonald, M.D.*

Moyer, John H., and Ackerman, Alfred J. Sarcoidosis: clinical and roentgenological study of twenty-eight cases. *Am. Rev. Tuberc.*, March, 1950, *61*, 299–322.

This is a clinical and roentgenologic study of 28 cases of sarcoidosis. A few cases were followed for as long as five years but no case was followed to death. Case reports are selected to illustrate the manifestations of involvement of mediastinum, lung, heart, bone, lymph nodes, liver, skin, parotid gland, eye, nervous system, and serous and mucous membranes. The authors feel that the final diagnosis of sarcoidosis rests on the histopathologic examination. The major differentiation is between Hodgkin's disease and tuberculosis. Other conditions to be considered and excluded are silicosis, lymphatic pulmonary carcinomatosis, pulmonary congestion, lymphosarcoma, leukemia, erythema nodosum, syphilis, fungus infection and bronchioitis. There is no known specific treatment for sarcoidosis. One of the cases in this group was proved to have active tuberculosis.—*V. P. Collins, M.D.*

Theodos, P. A., Gordon, B., Lang, L. P., and Motley, H. L. Studies in the clinical evaluation of disability in anthracosilicosis. *Dis. of Chest*, March, 1950, *17*, 249–272.

The authors present a thorough and detailed study of 375 hard-coal miners with various degrees of anthracosilicosis with special reference to respiratory impairment. The history, physical examination, exercise test, laboratory studies, electrocardiogram, chest roentgenography and fluoroscopy, bronchoscopy, bronchography and pulmonary function tests are included in the studies.

Cardiovascular diseases were found in 18.4 per cent, right heart strain being present in 16.2 per cent, this having a definite relationship to the degree of emphysema. Pulmonary tuberculosis was present in 12.5 per cent, nonspecific secondary infections in 12.5 per cent, arterial hypertension in 10.1 per cent, bronchiectasis in 3.4 per cent, senility in 3.5 per cent and syphilis in 3.2 per cent. Nineteen other conditions are listed as occurring less frequently. There were 6 cases of bronchogenic carcinoma (1.6 per cent). In 186 cases (49.5 per cent) the disability was complicated by one or more infectious, metabolic or degenerative conditions sufficient to cause work stoppage. In the remaining 189 (50.5 per cent) silicosis was regarded as producing the main disability on a pulmonary basis.

The impairment of pulmonary function was evaluated in 150 of these cases and correlated with the clinical impression. The main complaint was dyspnea, being present in 86 per cent; in about 60 per cent of these, the symptom was present for a period of three years or more before the disability necessitated work stoppage. In making a correlation between the predicted and the actual impairment it was found that in only 49 per cent of the cases were the predictions correct.

The factors reducing the arterial oxygen saturation in anthracosilicosis are grouped as follows: (1) ventilation: decreased maximum breathing capacity and vital capacity, (2) emphysema: increased residual air with the ratio of the residual air to the total volume increased, (3) ventilation efficiency: decreased oxygen uptake and carbon dioxide addition to the air breathed, and (4) distribution: unequal

alveolar aeration and perfusion. These factors may be involved in varying degrees and combinations with the result that a clinical estimation of the functional impairment becomes very difficult. Only marked decreases in ventilation and pronounced degrees of emphysema can be recognized clinically. With the use of fluoroscopy and the roentgenogram the accuracy is increased; the degree of emphysema can be predicted in about 50 per cent of the cases, this percentage being somewhat greater in advanced cases. When the disturbance, however, is mainly in the respiratory gas exchange the degree of functional impairment cannot be estimated from clinical examination. Likewise, little relationship was found to exist between the degree of functional impairment and the stage of silicosis or the length of exposure.—*J. P. Bennett, M.D.*

Forsee, J. H., Mahon, H. W., and James, L. A. Cavernous hemangioma of the lung. *Ann. Surg.*, March, 1950, *131*, 418–423.

The authors present 1 case of cavernous hemangioma of the lung, with detailed laboratory, roentgen, and surgical findings. Roentgen studies, including an angiogram, and the surgical specimen are illustrated. Following the case presentation, there is a short discussion of the anatomy and physiology of the defect and its effect on the patient (characteristic symptomatology). This case had originally been diagnosed and treated as pulmonary tuberculosis. The patient was cured by lobectomy.—*K. F. Kesmodel, Jr., M.D.*

Hochberg, L. A., and Pernikoff, M. Primary chondromas of the lung. *Dis. of Chest*, March, 1950, *17*, 337–346.

Primary chondroma of the lung is reported as a rare tumor with an incidence of less than 0.05 per cent. It is found most commonly in the age group of sixty to seventy years. It is separated from hamartoma on the basis of having a predominance of cartilaginous tissue with only minimal amounts of fibrous or osseous tissue. The generally accepted theory of origin is that it arises in embryonic rests. The authors add 3 cases to the 75 already reported, making a total of 78 cases.

The authors noted that there are no specific criteria for the clinical diagnosis of this tumor. It occurs in any part of the lung field and the symptoms and signs produced depend on the physical characteristics of the tumefaction—size, location, duration and secondary effects upon the surrounding structures. Many are asymptomatic.

Roentgenographically chondroma of the lung appears as a round or ovoid mass, somewhat nodular in outline, well delineated from the surrounding lung tissue and sometimes containing patchy areas of greater density.

The authors feel that since the clinical diagnosis is not always definite and the tumor may undergo sarcomatous changes, surgical removal is indicated.—*W. S. Fee, M.D.*

Bragg, E. A., Jr., and Levene, G. Hamartoma of the lung. *Radiology*, Feb., 1950, *54*, 227–235.

Albrecht, in 1904, proposed the term "hamartoma" for a benign mixed tumor occurring in various organs including the lung. Hamartomas are tumor-like malformations due to abnormal mixing or development of the normal tissue components of the organ. About 100 cases of pulmonary hamartoma are recorded in world literature. Occasionally the lesion is incorrectly diagnosed as chondroma.

Hamartoma of the lung is probably only second to bronchogenic carcinoma in occurrence.

The gross and microscopic picture is described.

The following are the usual findings of hamartoma:

1. Sharply defined tumor in the lung parenchyma with clear lung tissue surrounding it.
2. Lobulation of the margins in most cases.
3. Peripheral or subpleural location usually.
4. Irregular patches of calcification in many cases.
5. Areas of lesser density near the periphery of the tumor in some cases, due to collection of fat.

The authors describe differentiation from bronchogenic carcinoma, solitary metastasis, bronchial adenoma, tuberculosis and echinococcus cyst.

They report 3 cases discovered in routine chest examination and proved by surgery.—*E. E. Seedorf, M.D.*

Sheinmel, A., Roswit, B., and Lawrence, L. R. Hodgkin's disease of the lung; roentgen appearance and therapeutic management. *Radiology*, Feb., 1950, *54*, 165–179.

In order to demonstrate the variability of the pulmonary manifestations of Hodgkin's disease the authors selected 35 proved cases in

which the roentgen findings simulated other diseases.

When the disease apparently involves the lungs and mediastinum alone, the diagnosis is quite difficult but this is infrequent. No claim is made for pathognomonic changes but the cases discussed merely represent all the various types of pulmonary involvement in Hodgkin's disease. Lobular infiltration was the most frequent manifestation in this series while in 17 instances the changes were identical with metastatic malignancy from the thyroid, testes, or kidneys. A few cases simulated bronchogenic carcinoma and tuberculosis.

It is believed that the development of pulmonary involvement in Hodgkin's disease gives a poorer prognosis than when the disease involves lymph nodes alone, but there is no apparent correlation between the type of pulmonary involvement and the response to treatment. The use of roentgen therapy and nitrogen mustard is discussed.—*C. A. Stevenson, M.D.*

Bonoff, Chester P. Acute primary pulmonary blastomycosis. *Radiology*, Feb., 1950, *54*, 157–164.

Twenty-three cases of acute primary pulmonary blastomycosis constitute the body of this report. These patients were members of the U. S. Army located on the island of Okinawa during World War II. Six patients showed positive chest roentgenograms and are presented in detail.

This disease is similar to other pulmonary fungus infections and is characterized by chest pain, cough and moderate elevation of temperature and white blood cell count. The organism can be isolated from the sputum in all cases and is the only reliable diagnostic finding. No specific roentgen findings are present but the pulmonary changes consist of hilar adenopathy and patchy infiltration in the lung fields.—*C. A. Stevenson, M.D.*

Stein, Joseph, and Sheinmel, Archie. Cavitary disease of the lungs (due to less frequent etiological factors). *Radiology*, Feb., 1950, *54*, 219–226.

Pulmonary cavitation is most commonly seen in tuberculosis. Other causes for cavitation are aspiration pneumonitis, septic emboli, nonspecific emboli which become infected, mycotic disease, foreign bodies in the bronchi or lung parenchyma, benign and malignant neoplasms. The authors present 5 cases with cavitation traceable to infrequent causes including Hodgkin's disease, coccidioidomycosis, aspergillosis, foreign body and adenoma. Histories, progressive roentgenograms, and differential diagnostic problems accompany each case.—*E. E. Seedorf, M.D.*

Collins, Martha D., and Fisher, Hyman. A case of generalized hemangio-sarcomatosis erroneously considered as generalized tuberculosis. *Am. Rev. Tuberc.*, Feb., 1950, *61*, 257–262.

A twenty-five year old white male with chest pain and cough and blood-streaked sputum rapidly developed a pleural effusion, pericardial effusion, soft irregular mottling in both lungs consistent with miliary tuberculosis, and swelling in the left neck. Three sputum smears were positive for acid-fast bacilli, but bacteriological proof was not obtained.

He died six months after the onset of symptoms. Autopsy revealed a generalized hemangiosarcomatosis with the primary tumor in the deep tissues of the neck. Grossly and histopathologically the tumor resembled a Kaposi sarcoma. The only evidence at necropsy of tuberculosis was a small, healed primary complex in the upper lobe of the left lung.—*Lois C. Collins, M.D.*

Buckles, Maurice G., and Neptune, Wilford B. Tuberculous bronchitis in pulmonary resection; incidence in fifty-eight pulmonary resections for tuberculosis. *Am. Rev. Tuberc.*, Feb., 1950, *61*, 185–192.

The surgical specimens of 58 cases of pulmonary resection for tuberculosis revealed a marked discrepancy between bronchoscopic and pathologic diagnosis of tuberculous endobronchial disease. Only 8 (14 per cent) cases were positive at bronchoscopy just prior to surgery, but 38 (66.6 per cent) had microscopic evidence of tuberculous bronchitis in the resected specimen. There was no relationship between the extent of the disease and the incidence of endobronchial disease. However, in this series of resections only 3 cases were minimal in extent.

The high incidence of endobronchial disease probably accounts for failure of pneumothorax in many of these patients prior to surgery. It also seems likely that it accounts for some of the "spreads" which occur in the late postoperative period of some resections for tuberculosis.—*Lois C. Collins, M.D.*

Hamil, Brenton M. Bronchopulmonary mycosis; simultaneous primary occurrence in 4 children and their mother with subsequent healing by diffuse miliary calcification; a 12-year observation. *Am. J. Dis. Child.*, Feb., 1950, *79*, 233–271.

The importance of fungi as causes of pathologic changes in the lungs associated with acute and chronic respiratory illnesses is generally recognized. Their relation to pulmonary calcification not proved to be tuberculosis is an important and disputed question. The fewness of cases of pulmonary mycoses reported in children, especially within family groups, the author feels, justifies presenting the record of 5 cases of bronchopulmonary mycosis in a single family.

Bronchopulmonary mycosis (due to *Monilia pinoyi* and *Aspergillus niger*) developed simultaneously in 4 children and their mother with symptoms characteristic of and diagnosed as "la grippe" or "flu." The incriminated organisms were probably in the respiratory tract from a common source in nature (grain and hay dust and contaminated chicken feathers) and became pathogenic as a result of this conditioning illness. The subsequent course of the disease in the children was protracted because of inadequate early treatment. Massive doses of iodides (potassium iodide orally, sodium iodide intravenously and inhalations of ethyl iodide) were followed by progressive healing. The fluffy parenchymal nodules, characteristic of the roentgenograms in all patients, healed through fibrosis to scattered, complete, dense miliary calcification. The first appearance of calcium in all patients was about three years after the onset of acute symptoms. Serial blood counts and sedimentation rates showed characteristic changes similar to those observed in tuberculosis. Repeated tests of skin reaction to intradermal injection of various stock fungi and bacterial vaccines and to autogenous *Monilia pinoyi* and *Aspergillus niger* indicated the extent of allergic response to heterologous fungi but also showed the more specific response to the autogenous organisms. Complement fixation and agglutination reactions are given in detail in the paper.

Serial roentgenograms of the 4 patients are reproduced showing the progressive changes over ten years of observation.—*R. S. Bromer, M.D.*

Schmidt, Herbert W., Clagett, O. Theron, and McDonald, John R. Bronchiolithiasis. *J. Thoracic Surg.*, Feb., 1950, *19*, 226–245.

The literature on this subject is reviewed and 41 cases seen at the Mayo Clinic are presented.

The most common symptoms are: cough (often productive), hemoptysis, fever and pleuritic pain. Many cases give a history of having coughed up one or multiple stones.

The most important diagnostic finding is the presence of a dense area of calcium deposit at the apex of a triangular portion of collapsed lung. Pulmonary abscess or empyema may develop. If the broncholith is small, nothing abnormal may be seen or only multiple areas of calcification throughout both lungs or unilateral or bilateral hilar calcifications without evidence of collapsed lung may be present. Sometimes the picture may be very suggestive of bronchiogenic carcinoma.

Most cases bronchoscoped were considered to have carcinoma prior to bronchoscopy. The findings were often at first suggestive of tumor since frequently the broncholiths may occur in association with carcinoma. The most important factors in the production of broncholith are (1) calcareous or osseous change in peribronchial lymph nodes subjacent to larger bronchi (due to tuberculosis, coccidioidomycosis or histoplasmosis); (2) calcareous or osseous changes in bronchial cartilages as commonly seen in old age or following inflammatory processes. The lymph nodes may ulcerate into a bronchus and become detached. The peripheral lung may show "obstructive pneumonitis." The right lung is involved more often than the left, due to the greater number of lymph nodes associated with the right lung. The problem of a peripherally placed indeterminate bronchial lesion presents itself if the broncholith is located in a part of the bronchial tree that cannot be visualized by bronchoscopy. Surgical exploration must be undertaken in these cases.—*F. M. Reis, M.D.*

Erber, Bruno. Endobronchite tubercolare e fisiopatologia delle caverne. (Endobronchial tuberculosis and the physiopathology of cavities.) *Arch. di chir., ortop., e di med.*, March-April, 1950, *15*, 153–169.

Every tuberculous cavity has a drainage bronchus. Endobronchial tuberculous processes such as inflammatory swellings, scars, or plugs of blood or other secretions can interfere with the bronchial functions of ventilation and drain-

age and influence the course of the cavity. Obstructing ventilation could cause ballooning, increased intracavitary pressure, and rapid changes, sometimes with complete obliteration of the cavity. Obstructing drainage could cause partial or complete filling of the cavity by retained secretions. When the endobronchial process is an ulcer or similar lesion not reducing the lumen, none of the above changes occur.

The volume, contour and course of a cavity is largely determined by the intracavitary pressure which in turn is influenced by endobronchial changes, pericavitary atelectasis, intraalveolar pressure, masses within the cavity, and the elastic tension of the lungs, diaphragm and thorax.

Occlusion of the drainage bronchus is of primary importance in the healing of a cavity since it causes absorption of the intracavitary gases with subsequent reduction of oxygen, thereby inhibiting the growth of the tubercle bacillus.

Numerous factors may impede closure of the cavity and are merely mentioned: retained secretions which become infected, pericavitary atelectasis generally attributed to a complex neuromuscular reflex of the bronchial tree, fibrosis of the walls of the cavity and the surrounding lung which impairs absorption of gas due to a deficient blood supply, extensive fibrocaseous disease prohibiting lung retraction, pleural adhesions, collateral ventilation of the cavity and stenosis of the drainage bronchus.

Cavities may heal by open intervention, or by fibrosis and calcification which in the latter instances usually follow bronchial occlusion. A cavity with an occluded bronchus which fills with secretions may also heal by fibrosis and calcification although at times a central cold abscess may remain as a residue.

Treatment is aimed at the production of bronchial occlusion by collapse measures, or the evacuation of the cavity contents by Monaldi suction or by bronchoscopic aspiration in certain cases involving the lower lobes.—*C. Colangelo, M.D.*

Michelis, Ugo. La tubercolosi dell'albero bronchiale. (Tuberculosis of the bronchial tree.) *Arch. di chir., ortop., e di med.*, March-April, 1950, *15*, 121–152.

Tuberculous bronchitis produces symptoms of itself, but it is also important because of the changes it causes in both diseased and normal lung as the result of stenosis, occlusion, inflammation, ulcer or fistula. Thus, there may be dissemination of organisms into uninvolved lung segments, or there may be interference with drainage and/or ventilation, giving rise to a wide range of symptoms and findings. There may be intermittent retention of secretion causing bouts of fever and cough, fluid level in a cavity, ballooning of a cavity, rapid change in cavity size, including disappearance changes in intracavitary pressure both above and below atmospheric pressure, localized emphysema, atelectasis, pendulum movements of the mediastinum, lung abscess, bronchiectasis and massive collapse of the lung on attempting pneumothorax.

Tuberculous bronchitis may occur as part of the primary complex. Bronchial obstruction caused by granulation tissue, secretions or the pressure of large lymph nodes produces atelectasis which is a prominent feature of epituberculosis. Large glands may also erode into adjacent bronchi causing fistulae. Symptoms are often minimal while the roentgen findings consist of mottled, sometimes confluent opacities usually in the lower lobes, hilar adenopathy, and narrowed irregular bronchi on tomograms.

Tuberculous bronchitis as found in adult tuberculosis may be found anywhere, but is often seen in apical lesions whose bronchi drain into the hilum. It may exist independently after the lung lesion has healed. Tomograms may show narrowed, irregular, stenotic bronchi or bronchi with an abnormal configuration, while the lesion may be seen by bronchoscopy.

Bronchial lesions influence parenchymal disease. With obstruction of drainage, secretions are retained, the disease is aggravated, and atelectasis is likely. Atelectasis predisposes to infection, to increase in cavity size and to the formation of new cavities. If there is total bronchial occlusion, when the intracavitary gas is absorbed, the cavity may disappear.

The symptoms and findings include wheezing, rhonchi, whistling rales, cough, retrosternal pain, dyspnea, cyanosis, and fever.

Therapy includes rest, postural drainage, streptomycin both aerosol and parenteral, cough mixtures, etc. Through a bronchoscope one may use silver nitrate cautery, vasoconstrictors, dilating bougies, etc., but the results are variable.

Intrapleural pneumothorax is confined to mild lesions which do not threaten bronchial obstruction. Complications are frequent and militate against this method as well as against

extrapleural pneumothorax and phrenic intervention.—*C. Colangelo, M.D.*

Ariel, I. M., Avery, E. E., Kanter, L., Head, J. R., and Langston, H. T. Primary carcinoma of the lung; a clinical study of 1,205 cases. *Cancer*, March, 1950, *3*, 229–239.

The authors reviewed 1,205 cases of primary carcinoma of the lung seen from 1931–1946. The average age of the patient was 43.2 years. Other series indicate 78.9 per cent preponderance in males. The right lung was more often involved than the left (60 per cent).

The initial symptom was most often chest pain while cough was the next in frequency. Dyspnea, fatigue, and hemoptysis followed in that order.

Various diagnostic methods were mentioned. Cytological examination of bronchial secretions seems promising. The authors do not favor the aspiration biopsy through the thoracic wall because of the possible spread. They do recommend exploratory thoracotomy if clinical symptoms suggest malignancy.

Untreated patients have an average duration of life (from onset of symptoms) of 9.4 months.

Surgery is thought to be the treatment of choice. However only 13.4 per cent were operated upon of which only 4.6 per cent were resectable. Since 1940 the figures suggest 40 per cent operability and 22 per cent resectability. This would still leave the majority for other types of treatment. Contraindications to surgery were poor general condition, involvement of the trachea, or presence of distant metastases. Average duration of life following surgery was fourteen months. Five year survival was 7.1 per cent. If the patient survived the first year, he seemed to have a good chance of surviving four years and possibly longer.

Roentgen therapy with 250 kv., 2 mm. Cu., 50 cm. target skin distance, was given through four ports 10 by 10 cm. in size. A daily dose of 150 r to two parts was given until a tumor dose of 3,000 r was reached. The average survival time from onset of symptoms was 12.8 months. Those who received between 5,000 r and 6,500 r (in air) as a total dose appeared to have the best survival rate.

Teleradium, interstitial radium irradiation, and nitrogen mustard appeared to be much less satisfactory than deep roentgen therapy.—*R. C. Datzman, M.D.*

Heaton, T. G., and Ogden, W. E. Re-expansion of the artificial pneumothorax lung. *Dis. of Chest*, March, 1950, *17*, 298–303.

The authors report on the immediate results obtained in an analysis of the re-expansion period of 142 pneumothoraces at Western Hospital Chest Clinic. Thirty-six per cent were discontinued because of various complications before the operator would have chosen to permit re-expansion. The most common cause (16 per cent) was adhesive pleuritis; most of these cases, however, were close to the time at which discontinuance would have been chosen even without the complication. Ninety-one, or 64 per cent, were discontinued optionally. Of these, 44 per cent revealed a return practically to normal, i.e., without roentgen evidence of mediastinal shift or pleural thickening or fluid. The remainder all showed some evidence of these complications, these being present more frequently in those cases which had had a long duration of pneumothorax (over sixty months).

It would seem that a prolonged period of pneumothorax reduces the chances of full re-expansion. Other factors, however, must be considered since those cases with extensively diseased lungs are those which ordinarily are kept collapsed the longer. The authors give some evidence to indicate that pleural effusion prior to re-expansion also increases the chance of pleural thickening and mediastinal shift and hence should be prevented when possible. The authors are convinced that high negative pressures in the pleural cavity tend to promote a pleural effusion as a transudate. To prevent this it is suggested that refills be made at frequent periods (even at weekly intervals if needed) where the tendency of the lung to re-expand is impaired. It is also suggested that re-expansion be gradual, i.e., that the pneumothorax refills should not be stopped suddenly.—*J. P. Bennett, M.D.*

Brewer, Lyman A., Dolley, Frank S., and Evans, Byron H. The surgical management of chronic "spontaneous" pneumothorax. *J. Thoracic Surg.*, Feb., 1950, *19*, 167–198.

Spontaneous pneumothorax is not "idiopathic" for, in the great majority of cases, the cause may be found in (1) congenital cysts, (2) localized acquired subpleural blebs or bullae, and (3) acquired blebs or bullae, part of a more generalized pulmonary emphysema. The etiology of these lesions is discussed. Failure of the collapsed lung to re-expand results in chronic invalidism for the patient. The four important

factors in the persistence of the pneumothorax are the epithelial lining of congenital cysts, the presence of intrapleural adhesions, the scar tissue in the lung or bronchiole, and the formation of an inelastic visceropleural membrane. The presence of this fibroplastic membrane covering the lung is reported here for the first time and its surgical importance is stressed.

The authors present 15 cases which were treated by the following procedures: (1) complete pneumonolysis, (2) surgical closure of the pulmonary openings, (3) resection of pulmonary cysts or bullae, and (4) decortication of the lung. The only other therapy has been the production of a chemical pleurodesis by virulent pleural irritants. This method of therapy is definitely inferior to the surgical treatment, because of the limitation of pulmonary function so produced.

Roentgenograms usually fail to show the cause of the pneumothorax. It is important to rule out the presence of a so-called symptomatic pneumothorax. Giant cysts of the lungs may simulate a spontaneous pneumothorax. Evidence of trabeculae in the cysts or loculations of the cysts are important diagnostic aids. In some instances the visceral pleural membrane shows up as a thickened pleural shadow.—*F. M. Reis, M.D.*

OSWALD, NEVILLE. Pulmonary changes in the reticuloses. *Proc. Roy. Soc. Med.*, March, 1950, *43*, 208–213.

Hodgkin's disease, lymphosarcoma, lymphatic leukemia, mycosis fungoides, chronic myeloid leukemia, acute leukemia, disorders in lipoid metabolism including Gaucher's disease, Niemann-Pick's disease and the triad of Letterer-Siwe and Hand-Schüller-Christian disease and eosinophilic granuloma of bone are included in this group, also leukemoid states.

The lungs with their abundance of reticuloendothelial cells and rich blood supply will often be affected in such disorders as the reticuloses.

In Hodgkin's disease paratracheal, tracheobronchial and bronchopulmonary groups of lymph nodes may be affected with resulting pressure effects that may lead to collapse of lung, edema and/or of pleural effusion. Parenchymal infiltration may represent blood-borne metastases or infiltration may resemble pulmonary tuberculosis.

The triad of lymphosarcoma, lymphatic leukemia and mycosis fungoides are closely related to lymphadenoma in their manifestation in that enlarged mediastinal nodes or pleural effusions are the main manifestation. When the lungs themselves are invaded the roentgen appearances are indistinguishable from each other or from Hodgkin's disease. The rate of progress of the latter triad tends to be rather slower.

Pulmonary changes in chronic myeloid leukemia are usually slight and inconclusive. On occasion slight enlargement of mediastinal nodes may occur. Rarely there is diffuse infiltration of the lungs with fibrosis and infarction.

In approximately one-third of the cases of acute leukemia pulmonary lesions are dramatic and comprise pneumonia, infarction, edema and infiltration of immature cells. Partial and complete thromboses are common, most larger lesions have started as infarcts. These occasionally break down and form cavities which often give little in the way of symptoms.

In the disorders of lipoid metabolism pulmonary findings are rare in Gaucher's disease. The manifestations in Niemann-Pick's disease are usually edema and congestion. In Letterer-Siwe disease, Hand-Schüller-Christian disease or eosinophilic granuloma of bone, pulmonary findings may be quite similar regardless of which condition exists elsewhere and are different from anything seen in any of the other diseases of the reticulo-endothelial system. The lesions are diffuse throughout both lungs. Early there is an infiltrative appearance suggestive of reticulo-endothelial cell proliferation. This in turn may lead to fibrosis and cyst formation giving a honeycomb appearance.

Leukemoid states may be provoked by pneumonia such as occurs in infants and young children at times; whooping cough and occasionally pulmonary tuberculosis may be associated with leukemoid response.

The article is illustrated with reproductions of chest roentgenograms of different types of pulmonary manifestations in the reticuloses and there is one table showing leukemoid reactions during a terminal case of miliary pulmonary tuberculosis.—*J. H. Gilmore, M.D.*

LEVINSON, DAVID C., EDMEADES, DONALD T., and GRIFFITH, GEORGE C. Dissecting aneurysm of the aorta: its clinical electrocardiographic and laboratory features; a report of fifty-eight autopsied cases. *Circulation*, March, 1950, *1*, 360–387.

Fifty-eight autopsied cases are analyzed; 16 of these cases were diagnosed antemortem.

Thirty-six cases occurred between the ages of fifty and seventy. The incidence in men was 26 times that in women. Fifteen cases were in Negroes. Clinically the cases are described as acute (death within forty-eight hours), subacute (symptoms over days or weeks), and chronic (survival of initial dissection).

Sudden severe tearing pain not usually related to effort and usually referred to chest or epigastrium or both was the presenting symptom in 77.6 per cent of cases. Radiation of pain may occur to back or extremities. Recurrent pain occurred in 50 per cent of the cases. Syncope was rare. Six patients were unconscious. In these cases the dissection involved major blood vessels to the brain. Dyspnea was frequent. Vomiting occurred in about 20 per cent of the cases. Clinically, the patient was in acute distress with pulse rate usually elevated and bradycardia occasionally. Temperature elevation was found in about half the cases on admission while the others who survived developed an elevated temperature. Hypertension was present in 56 per cent. Eleven cases in addition showed left ventricular hypertrophy.

Pleural fluid was found in 6 cases. Cardiac enlargement was common. Pericardial hemorrhage was found at autopsy in 34 per cent, but was not suspected antemortem; pericardial friction rub was rare. Cardiac murmurs were heard in 32 cases. "The sudden appearance of a basal aortic diastolic murmur in a patient with hypertension complaining of severe chest or abdominal pain is almost pathognomonic of a dissecting aneurysm of the aorta."

Pulsating masses were found in 4 cases and vessel tenderness in 2 cases. Eleven patients showed unequal or absent pulsations of peripheral arteries. Nine patients presented neurologic findings of varying degrees.

Electrocardiograms were made in 40 cases. The patterns were all abnormal, but not diagnostic of dissecting aneurysm.

Roentgen examination, usually by portable film, showed widening of the supracardiac shadow in 10 of 20 patients examined. Serial films may show increase in width of the aorta. Cardiac enlargement is shown in over half the cases. Pleural and pericardial effusion may be found.

Positive Wassermann or Kahn tests were found infrequently. A normochromic normocytic anemia was frequent. There was a moderate to a marked leukocytosis. Albuminuria of varying degrees was common. Hematuria occurred in 8 cases. Non-protein nitrogen was elevated in 7 of 9 patients examined. Serum amylase was elevated in 2 cases.

In summary: the patients usually have a history of hypertension; the onset is sudden, with shock; neurologic symptoms may occur; aortic diastolic murmurs occur in a fourth of the cases; inequalities of pulse are important; electrocardiogram shows left ventricular strain; roentgen examination shows wide aortic shadow; laboratory data may help.

The prognosis is poor and the treatment is symptomatic.—*E. C. Baker, M.D.*

Zimmerman, Henry A., Scott, Roy W., and Becker, Norman O. Catheterization of the left side of the heart in man. *Circulation*, March, 1950, *1*, 357–359.

Catheterization of the left side of the heart is difficult. Retrograde passage of the catheter against arterial pressure, arterial spasm, the very short interval of open aortic valves, the unknown position of valves, eddy currents, and the negative pressure when the jet of blood is stopped, all contribute to the problem. Catheterization of the left ventricle has only been successful in cases of aortic insufficiency.

The catheter is introduced through the left ulnar artery and passed under fluoroscopic guidance. Heparin solution is given through the catheter. The passage of the catheter must be quick and smooth. In 11 cases with syphilitic aortic insufficiency, the left ventricle has been catheterized successfully. One case of rheumatic aortic insufficiency had an abrupt appearance of ventricular fibrillation which could not be relieved. This was apparently caused by the catheter blocking an anomalous coronary artery.—*E. C. Baker, M.D.*

Heberer, G. Zum Krankheitsbild des Pneumoperikards. (The disease process of pneumopericardium.) *Chirurg*, March, 1950, *21*, 174–177.

Pneumopericardium, secondary to trauma, tumors and infections adjacent to the heart, or septicemia due to anaerobic organisms, is rarely seen. The author discusses an unusual sequence of events and presents roentgenograms illustrating the various complications of the disease in an eight year old boy.

The course was as follows: left lower lobe pneumonia; left-sided pneumococcal empyema (after three weeks); pyopneumothorax (after thoracocentesis), requiring subsequent insertion of a drainage tube; pyopneumopericardium (by direct extension from the empyema);

pneumopericardium (due to spontaneous replacement of fluid in the pericardial sac by air from the pleural cavity); death, three days later, as a result of cardiac tamponade and toxemia.

Autopsy, eight weeks after onset of illness, showed evidence of a pre-existing, fibrinous pericarditis. It was therefore, postulated that a locus minoris resistentiae existed in an area of pleuropericardial adhesions; furthermore, that the drainage tube had been inserted too close to the vulnerable pericardial sac, thus facilitating perforation of the latter.

The diagnosis of pneumopericardium is suggested by the clinical finding of tympany over a tent-like, widened precordial area. The roentgenogram is unquestionably diagnostic and easily excludes pulmonary cavitation, pneumothorax, mediastinal emphysema, etc. Small amounts of air in the pericardial sac may be spontaneously absorbed but aspiration is urged in tension pneumopericardium to avoid the danger of cardiac tamponade.—*J. Zausner, M.D.*

Wood, Paul. Cardiac complications (discussion of the management of rheumatic fever and its early complications). *Proc. Roy. Soc. Med.*, March, 1950, *43*, 195–199.

The authors state that approximately 5 per cent of the British population are known to have acute or subacute rheumatism before they are twenty years of age. Therefore in any patients with active rheumatic fever or suspected rheumatic fever it is vitally important to detect the presence or absence of clinical carditis for proper, immediate and future management depends upon the accuracy of such a diagnosis.

The chief signs of active carditis are as follows: (1) significant mitral systolic murmur; (2) the Carey Coombs murmur; (3) a new or transient aortic diastolic murmur; (4) cardiac enlargement; (5) pericardial pain, friction or effusion; or electrocardiographic proof of pericarditis; (6) elevation of the jugular venous pressure and other evidence of heart failure; (7) prolongation of the P-R interval or of Q-T_c by electrocardiography.

Roentgenological information has been disappointing in detecting the presence of carditis. Cardiac enlargement is unusual unless the heart is in failure, there is pericardial effusion, or old valvular lesion. Slight reduction in heart size may follow subsidence of the fever. Other things being equal, the diastolic heart size is apt to be larger when the rate slows and with the short exposure time in common use today series of films may show heart in systole or diastole and may be a simple trap for the unwary.

Roentgenograms were used in detection of pericardial effusion by making films in prone or supine position and revealing widening of the vascular pedicle with disappearance of the first inch of the descending aorta and divergent vascular shadows near the thoracic inlet. These signs have not been reliable thus far. Catheterization of the right atrium has been used and in pericardial effusion the right border formed by the parietal pericardium may lie well beyond the tip of the catheter. Angiocardiography offers an alternative means of providing similar information but is hardly justified in these sick children. The cardiac catheterization is far less unpleasant and is well tolerated.

Expansile pulsation of the left auricle is seen in a small minority of cases of active pericarditis with loud mitral systolic murmur. Advanced organic mitral incompetence presents two main forms: (1) with left ventricular enlargement which is apt to be confused with aortic stenosis; (2) with aneurysmal dilatation of the left auricle.

The article is illustrated by reproductions of roentgenograms of various heart shapes. Reproductions of electrocardiographic tracings are included.—*John H. Gilmore, M.D.*

Abbott, O. A. Congenital aneurysm of superior vena cava; report of one case with operative correction. *Ann. Surg.*, Feb., 1950, *131*, 259–263.

The author reports on the diagnosis and treatment of a large, probably congenital, aneurysm of the superior vena cava which was discovered roentgenologically in a nineteen year old male college student. The mass which protruded to the right of the superior mediastinum had a sharp lateral outline, was situated anteriorly and changed shape on inspiration and expiration. It was further studied by means of angiocardiographic techniques using 70 per cent diodrast and cardiac catheterization. The mass increased in size over a two year period and the patient was operated upon at which time the presence of the vena cava aneurysm was confirmed. It was treated by enclosure with polythene cellophane. The

patient has done well postoperatively.—*C. L. Hinkel, M.D.*

Epstein, B. S. Angiocardiography; its application to the diagnosis of coarctation of the aorta. *Radiology*, Feb., 1950, *54*, 211–218.

This is a report of 1 adult and 2 children who had opacification studies with subsequent demonstration of coarctation of the aorta. The author recognizes the fact that all those interested in cardiovascular malformations do not think that angiocardiography is advisable or necessary in instances of coarctation; however, he states that the value of the test is in determining the exact location of the aortic narrowing, the size of the aorta above and below the coarctation and the degree of development of collateral circulation.—*C. A. Stevenson, M.D.*

Dow, J. W., Levine, H. D., Elkin, M., Haynes, F. W., Hellems, H. K., Whittenberger, J. W., Ferris, B. G., Goodale, W. T., Harvey, W. P., Eppinger, E. C., and Dexter, L. Studies of congenital heart disease. IV. Uncomplicated pulmonic stenosis. *Circulation*, Feb., 1950, *1*, 267–287.

The authors have made a study of 8 cases of uncomplicated pulmonic stenosis using cardiac catheterization along with the customary history, physical and roentgen findings. It was found that the systolic pressure was higher in the right ventricle than the pulmonary artery. This confirms the reports of two other authors using a recognized technique. The authors also review cases in the literature comparing their findings with those reported. The physical findings are those of a systolic murmur heard best in the 2nd, 3rd or 4th left interspace transmitted over the pericardium and back. The characteristic roentgenographic pattern is enlargement of the right ventricle and auricle if the stenosis is long standing. The electrocardiographic tracings are not characteristic except in 6 cases, consisting of right ventricular hypertrophy. The authors state that simple pulmonic stenosis cannot always be differentiated from pulmonic stenosis complicated with congenital defects unless cardiac catheterization is done. Those interested in circulatory dynamics of pulmonic stenosis should consult the article.—*C. H. Warfield, M.D.*

Bogoch, A., and Christopherson, E. F. Calcified cardiac aneurysms. *Ann. Int. Med.*, Feb., 1950, *32*, 295–308.

Cardiac aneurysms usually follow myocardial infarction; the left ventricle is almost exclusively affected. The diagnosis of cardiac aneurysm depends largely on roentgenologic examination which shows the heart usually enlarged to the left with a blunted apex; this gives the heart a square or rectangular appearance. Most characteristic finding is a localized bulge occurring anywhere in the contour of the left ventricle. Apical aneurysms may be picked up by fluoroscopic examination with the diaphragm fully depressed as in full inspiration.

Calcification in the wall of the cardiac aneurysm is a relatively uncommon occurrence, but may be of further aid in the roentgen diagnosis of cardiac aneurysm. This condition has to be differentiated from a calcified pericardium such as may occur in constrictive pericarditis. In this latter process the calcification produces a thicker and more irregular shadow not restricted to the left ventricle and situated outside the heart contour while in calcification of a cardiac aneurysm the calcified shadow is usually linear, limited to the left ventricle and situated within the heart contour.—*H. R. Fishback, Jr., M.D.*

ABDOMEN

Kondo, B., Winsor, T., Raulston, B. O., and and Kuroiwa, D. Congenital coarctation of the abdominal aorta, a theoretically reversible type of cardiac disease. *Am. Heart J.*, Feb., 1950, *39*, 306–313.

One of the rare (only 8 recorded cases in the last 115 years) conditions which may cause heart failure in a child is congenital coarctation of the abdominal aorta.

A twelve year old underdeveloped Japanese girl had complained of lassitude for three years, but there had been no definite cardiovascular symptoms. On examination, the heart was enlarged and the radial and femoral pulses absent. Auscultation demonstrated a systolic murmur with its maximum intensity in the region of the umbilicus. Bilaterally the seventh through twelfth intercostal arteries were seen to pulsate with each heart beat. Roentgenograms revealed an enlarged left ventricle and chronic passive pulmonary congestion. There was no rib notching. Right and left heart failure progressed and the patient died during an episode of pulmonary edema forty days after her first hospital admission.

Postmortem examination showed dilatation

of both ventricles and hypertrophy of the left ventricle; the heart valves, endocardium and coronary vessels were normal. The aortic arch was dilated and both subclavian arteries hypoplastic, the right subclavian being aberrant in origin. The wall of the dilated thoracic aorta was thickened and numerous atheromatous plaques were arranged along its intimal surface. A marked degree of coarctation of the abdominal aorta between the celiac axis and the renal arteries was present. A valve-like structure appeared at the level of the coarctation. Distal to the coarctation the aorta narrowed to about one-fourth the caliber of the thoracic aorta.

The diagnosis of congenital coarctation of the abdominal aorta during life should not be difficult—abdominal bruit, diminished femoral pulses, and hypertension in the upper extremities are suggestive. In coarctation of the thoracic aorta, notching of the third to tenth ribs may be present, while in the abdominal aorta coarctation the lower ribs (7th–12th) are more likely to be involved. Other conditions to be differentiated are embolus or thrombosis of the aorta and dissecting aneurysm. With correct early diagnosis surgical attack on this anomaly may become possible.—*Harold Fulton, M.D.*

HODGES, FRED JENNER. Downward displacement of the gastric cardia. *Radiology*, Feb., 1950, *54*, 194–201.

The gastric cardia, whether filled with gas or barium, lies closely applied to the under surface of the dome of the left diaphragm and maintains this position regardless of the position of the patient. Anteriorly the left lobe of the liver is interposed between the cardia and the left half of the diaphragm while posteriorly and laterally the spleen wedges between the diaphragm and the stomach.

The author urges that these anatomical relationships be kept in mind so that any downward displacement of the cardia can be recognized. Enlargement of the liver or spleen, intra-abdominal fluid, subphrenic abscess, and carcinoma of the cardia of the stomach are common causes for this displacement.—*C. A. Stevenson, M.D.*

EIBACH, ERNST. Kasuistischer Beitrag zur Gastritis polyposa. (Case report of polypoid gastritis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, March, 1950, *72*, 573–576.

Eibach reports a case of polypoid gastritis in a fifty-four year old male which he observed for over two years. The patient had complained of anorexia, abdominal pain without food relationship and had had a palpable mass in the epigastrium. Achlorhydria and secondary anemia were present; the latter improved during the period of observation. Occult blood in the stool was found on one occasion; tarry stools were not present. Roentgenologically the stomach had giant mucosal folds early during the period of observation, later reverting to normal and presenting a dilatation, without retention. The case is reported because of the associated anemia, which improved, and because of the lack of evidence of malignant degeneration during the period of observation. A review of the literature of the problem is also presented and illustrations of roentgenograms accompany the report.—*L. A. Nash, M.D.*

LADUE, JOHN S., MURISON, PAUL J., MCNEER, GORDON, and PACK, GEORGE T. Symptomatology and diagnosis of gastric cancer. *Arch. Surg.*, Feb., 1950, *60*, 305–335.

A study of 1,117 patients with gastric cancer was undertaken to determine whether any correlation existed between the duration and type of symptoms or physical findings and the resectability rate. The diagnostic and prognostic significance of gastric analyses, hematologic studies, stool examinations and gastroscopic and roentgenologic findings were evaluated in these patients. With the exception of dysphagia which was twice as common among patients with inoperable carcinoma, the frequency and multiplicity of symptoms of patients with inoperable and operable gastric cancer was essentially the same. The resectability rate for carcinoma involving the cardia of the stomach was 20 per cent greater than that for carcinoma involving the stomach as a whole. The symptoms presented by these patients in decreasing order of frequency were loss of weight, pain, vomiting, disturbances of the bowel, anorexia, dysphagia, nausea, weakness, eructation, hematemesis and rapid satiation. Loss of weight above 41 pounds was usually associated with gastric cancer in an operable stage but the rapidity of the loss as well as the percentage is more important than the absolute fall in poundage. Pain, dysphagia and anorexia were the first definitive complaints of the patients studied. Dysphagia was the first symptom experienced in 40 per cent

of the patients with involvement of the cardia. Pain occurred more prominently with cancers involving the lesser curvature of the body of the stomach. Pain caused by cancer of the cardia may simulate angina pectoris. Anorexia was more frequent with pyloric and prepyloric cancers. The duration of symptoms bore no relation to the resectability of the lesion. The surgical mortality was more than three times as great among patients whose symptoms had been present from eighteen to thirty-six months as among those whose complaints were of shorter duration. Fifty-three of 155 patients who had gastrointestinal symptoms for more than two years gave a history of ulcer-like pain; 102 had complained only of vague indigestion. In a large majority of the patients a change in symptoms developed which could have been an important clinical clue to development of carcinoma of the stomach. Mass roentgenologic studies do lead to early recognition of gastric cancer in from 0.2 to 0.5 per cent of persons without gastric complaints but at present these examinations are economically impractical. The presence of an epigastric mass does not indicate inoperability. An epigastric mass was found in 50 per cent of patients with inoperable and 42 per cent of patients with operable gastric cancers. A positive liver biopsy, peritoneal metastases and physical signs as signal nodes, Blumer's shelf, ascites, jaundice, nodular liver, pelvic implants interdict exploratory laparotomy.

Gastric analyses showed achlorhydria in 65 per cent of the patients with inoperable gastric cancer and in 51.1 per cent of those whose carcinomas were resected. Extensive carcinomas and anaplastic tumors showed a greater incidence of achlorhydria. Hyperchlorhydria was seen twice as frequently with benign gastric ulcers as with gastric cancer and three times as often in normal adults sixty to sixty-four years of age. Cytologic study of the gastric juice for cancer cells is an important additional diagnostic aid. The degree of anemia bore no relation to the segmental location of the gastric cancer or to its operability.

In 1,022 gastrointestinal studies of patients with gastric carcinoma a positive diagnosis was made in 91.5 per cent, an inconclusive diagnosis in 3 per cent and the findings were reported within normal limits in 5.5 per cent. By the employment of both roentgenologic studies and open tube gastroscope the incidence of positive diagnosis was increased from 85.1 per cent by roentgen examination alone to 96.3 per cent. Positive results of biopsy material obtained through the open tube gastroscope was secured in 71.4 per cent of the patients with accessible lesions. Examination with the flexible gastroscope in 144 patients gave positive evidence of cancer in 84.0 per cent and inconclusive evidence in 11.1 per cent, and was reported as showing no evidence of abnormality in 4.9 per cent. Results of roentgen examination were positive for 88.9 per cent of these patients and inconclusive for 9.0 per cent, and reported as within normal limits for 2.1 per cent. Gastroscopic and roentgenologic study of the same patients was confirmatory in 76.4 per cent. The incidence of positive diagnosis obtained by the use of both methods was 96.5 per cent—an increase of 7.6 per cent over the 88.9 per cent incidence of positive diagnosis obtained by roentgen examination alone. Among 305 patients who had both roentgenologic and gastroscopic examination, the use of the gastroscope raised the incidence of positive preoperative diagnosis 9.5 per cent. Abnormalities detected in the roentgenograms and/or gastroscopy and not diagnostic of cancer were found in an additional 3.3 per cent of 305 patients raising the probability of the presence of cancer in the stomach to 99.7 per cent.

Exploration should be done in every patient with persistent symptoms suggestive of carcinoma even where the laboratory and roentgen findings are negative.—*M. M. Friedman, M.D.*

Vogt, Alfred. Über das Verhalten der Häufigkeit des Magen und Zwölffingerdarmgeschwüres in den letzten zwölf Jahren. (The relationship of the frequency of gastric and duodenal ulcer during the past twelve years.) *Deutsche med. Wchnschr.*, March 17, 1950, *75*, 349–351.

The statistics summarized are from the author's Roentgen Institute of the Tübingen University and Polyclinic during 1937–1949. Several tables and graphed curves demonstrate various phases in the frequency of gastric and duodenal ulcers related to World War II. Until 1942 the proportion of patients with gastric ulcers, deformed duodenal bulbs and coarse gastric mucosal folds decreased, as did the number of patients with gastric complaints and of patients without positive roentgenologic findings. In contrast, the number of patients with florid duodenal ulcer increased sevenfold during this time. Then during 1943–1944, and until the end of the war in 1945, *all* these ab-

normalities increased in frequency. After some decrease, another increase in frequency occurred, related to the generalized want. Currently (in the first quarter of 1949) florid duodenal ulcer remains ten times as frequent as in 1937, and gastric ulcer two and a half times as frequent.

Duodenal ulcer was most common in the 21–30 year olds; gastric ulcer in the 41–50 year olds.—*H. G. Moehring, M.D.*

North, J. P., and Johnson, J. H., Jr. Pyloric hypertrophy in the adult. *Ann. Surg.*, March, 1950, *131*, 316–329.

Congenital pyloric hypertrophy has long been recognized as a distinct clinical entity in infants. It is not so generally realized that there may occur in adults a lesion which bears a striking resemblance to it. It is important that this disease be differentiated from other lesions at the pylorus because, although often disabling, it is benign and amenable to surgical correction.

Both the infantile and the adult forms of hypertrophy were noted at autopsy many years before they were accorded clinical recognition.

Secondary pyloric hypertrophy is commonly observed with both benign and malignant lesions of the stomach. In the primary hypertrophy discussed, no other gastric abnormality appears to be present.

The first surgical cure was reported in 1904. The authors collected 59 cases, with operative or necropsy verification, and added 5 of their own cases.

One-fourth of the patients had intermittent symptoms of pyloric disturbance, persisting throughout infancy and childhood into adult life. The more common history is that of a more or less abrupt onset of digestive disturbances, in a previously healthy, middle-aged adult. Runstrom found roentgenologic abnormalities long after active symptoms had subsided, in patients with congenital pyloric stenosis. This suggests that an organic basis may remain in unoperated patients as a potential source of trouble in adult life. Others favor an acquired etiology. Boerema found "many instances" of a rigid antrum in the Netherlands during the Nazi occupation. Hypertrophy of the pylorus was the only surgical finding. Nutritional deficiency was thought to be a contributory cause.

Roentgenographically, the lesion is hard to distinguish from an infiltrative neoplasm. Ordinarily, it is not confused with benign ulcerations. Delay in emptying is seen, but complete gastric dilatation is unusual. The pyloric canal is seen and is elongated. The narrow passage may be concentric or eccentric. Irregularities of mucosal pattern in the pyloric canal are due to projecting edematous folds of mucosa, and barium flecks are not retained. The changes persist after antispasmodic drugs. The mushroom deformity of the base of the duodenal bulb was not thought to be a reliable sign of pyloric hypertrophy by the authors.

At surgery, the stomach may present a tumor mass or thickening or seem normal. The surgeon is obligated to open the prepyloric portion of the stomach and inspect its interior. The pyloric ring should not be divided until after palpation has been done with a finger introduced through the pyloric canal. Once the sphincter is cut, it is very difficult to evaluate the situation.

The outstanding pathologic feature of this lesion is hypertrophy of the circular layer of muscle in the pyloric ring. Treatment is surgical. While lesser operations may suffice, the authors favor a limited gastric resection. When this is done, cure is certain and the distinction from carcinoma can be made readily.—*T. J. Wachowski, M.D.*

Shallow, T. A., Wagner, F. B., Jr., and Manges, W. B. Primary carcinoma of infrapapillary portion of duodenum. Surgery, March, 1950, *27*, 348–355.

The authors present a case of primary carcinoma of the infrapapillary portion of the duodenum in which the diagnosis was made preoperatively by roentgen methods. The lesion was segmentally resected and an end-to-end anastomosis of the duodenum and jejunum was done. The patient was well three years after the operation.

The condition is quite rare with only about 70 proved cases in the literature. The lesion was found at autopsy or was inoperable in four-fifths of the cases. Approximately 3 per cent of cases of intestinal carcinoma are in the small intestine and of these 37 to 45 per cent occur in the duodenum. The incidence is about the same in the three sections of the duodenum if the debatable tumors of the ampulla of Vater are excluded.

Fifteen cases of primary carcinoma of the infrapapillary portion of the duodenum have been reported that have had resections, including 2 cases of the authors. The 15 cases are listed, together with pertinent data including the roentgenologic diagnosis which was correct in 4 cases, incorrect in 3 cases, adequate in 2 cases, inadequate in 1 case and not done or mentioned in 5 cases. The difficulties in roentgen diagnosis of these lesions are mentioned, such as failure to look for or consider the lesion because of its rarity; if obstruction were present to diagnose a gastric neoplasm or ulcer; accurate localization was difficult if the site of the lesion was irritable; and terminating the examination upon finding pathology in the stomach or duodenal bulb. The lesion was well demonstrated roentgenographically in the case presented by the authors.—*C. W. Cooley, M.D.*

Pohlandt, Klaus. Detailstudien am pylorusnahen Duodenalabschnitt. (A detailed study of the juxtapyloric portion of the duodenum.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, March, 1950, *72*, 564–573.

Pohlandt presents a detailed study of the first portion of the duodenum together with a case presentation of a type of granular duodenitis (Schrotkornbulbus, i.e., "ground-meal duodenitis"). He discusses the sources of error in the diagnosis of deformity of the duodenal bulb, showing that the arcuate first portion may appear as having a definite eccentric deformity in the posteroanterior projection, but is entirely normal in the right anterior oblique position. The occurrence of a pseudo-crater in the distal portion of the bulb due to contrast medium in a mucosal fold in the second portion being projected through the bulb is described. The various types of mucosal patterns in the normal duodenal bulb are shown, which are essentially axial folds, oblique or diagonal folds or a reticular pattern. He reports in detail a case study with operative and pathologic findings of gastritis and duodenitis in a middle aged patient presenting abdominal pain, usually after meals with otherwise essentially normal findings. This he has described as a granular type of duodenitis with similar changes in the stomach, and is presented as a distinctive roentgenographic entity. Four sketches, nine illustrations of roentgenograms and microphotographs of the specimen accompany the presentation.—*L. A. Nash*, M.D.

Mordeglia, Miguel, Ferdman, José, and Entin, Juan. Preferente localización duodenal de una micosis fungoide linfosarcomatosa. (Preferential localization in the duodenum of a case of lymphosarcomatous mycosis fungoides.) *Semana méd.*, March 16, 1950, *57*, 383–391.

The authors describe a case of lymphosarcomatous mycosis fungoides in a forty-six year old man. The process began with skin manifestations in 1936 and was observed until the patient's death in 1948. In the course of the disease mediastinal and visceral tumors appeared of which the changes in the mediastinum and in the second portions of the duodenum were the most prominent. Considering the rarity of duodenal localization of the disease, the duodenal roentgen appearance (enlargement of the lumen, filling defects and rigidity of outlines) is described in detail. After very satisfactory results following the initial roentgen treatments, the lesions recurred and became refractory. Later therapeutic attempts with radioactive phosphorus and nitrogen mustard were unsuccessful. Of special interest is the fact that the tissue slides which were diagnosed as "mycosis fungoides" in Argentina were diagnosed as "reticulum cell sarcoma" in the United States. The authors stress the uncertainty in the histopathologic classification and the respective confusion in the medical literature regarding this clinical entity.—*Ernst Schmidt, M.D.*

Prévôt, R. Röntgendiagnostik der entzündlichen Dünndarmerkrankungen. (Roentgen diagnosis of inflammatory lesions of the small intestine.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, March, 1950, *72*, 547–563.

This is an extensive review of the literature of inflammatory lesions of the small intestine with presentation of illustrative cases. The author feels that it may be possible to differentiate the various inflammatory lesions of the small intestine with the same degree of accuracy as lesions of the stomach or colon.

He uses a fractional oral method of administration of the opaque meal, and studies the terminal ileum by the retrograde method.

The types of lesions discussed are acute, subacute and chronic enteritis, regional enteritis, non-sclerosing ileitis, necrotizing enteritis, tuberculosis and typhoid enteritis.

Acute enteritis is rarely studied roentgenographically. Subacute and chronic enteritis may be divided into the specific and nonspecific types. Mucosal swelling, induration of the wall and increased secretions occur. An example of this is seen in the jejunum following anastomosis to the stomach. The first stage of involvement by an inflammatory lesion is represented by loss of the serpentine mucosal pattern, the markings becoming more transverse. Patchy filling occurs because of the increased secretions. The second stage is represented by swelling of the mucosal folds, loss of the spaces between these folds and induration. The third stage seen postoperatively in the jejunum shows disappearance of the mucosal folds and narrowing of the lumen.

Non-tropical sprue is a special form of chronic enteritis differentiated by the lack of motility and puddling of the media. Atrophy of the mucosa may occur.

Regional ileitis may affect any portion of the small intestine including the duodenum. Clinically, the process presents itself as appendicitis or chronic ileus, with fever, diarrhea and palpable mass. Roentgenographically, there is evidence of an infiltrating process, narrowing the lumen of the bowel, and involving segments of the bowel, often quite long. Prestenotic dilatation occurs as does fistula formation between loops of bowel. The differentiation from tuberculosis is difficult and the relationship debatable.

Non-sclerosing enteritis described by Golden is discussed. Narrowing of the lumen occurs and polypoid changes in the mucosa are seen. This occurs in young females.

Necrotizing enteritis first described by Lubarsch during World War I was again seen in Germany in 1946 and 1947. This presented itself as a severe inflammatory process in the ileum and colon. Undoubtedly malnutrition played a part in its origin. Roentgenographically, the early findings are those of atony and ileus; later fractional filling occurs and the involved loops seem to be rigid and indurated. Loss of the mucosal pattern occurs and varying degrees of retention are also present. The lesions must be differentiated from tuberculosis and regional ileitis. This often has to be done clinically. Healing occurs without stenosis.

Tuberculosis enteritis is well known. The changes in the small intestine correspond to the ulceration, infiltration, polypoid hyperplasia, segmental involvement and adenomatous thickening of the wall. On examination eccentric or annular constrictions occur, the mucosa looks ragged and skip areas are noted. Irregularity of the lesions is a characteristic sign due to inflammatory tumefaction.

Typhoid and paratyphoid fever, while rarely investigated, involve short segments of the bowel and produce polypoid lesions, probably due to proliferation of lymphoid follicles and Peyer's patches.

Twenty-two illustrations accompany the article.—*L. A. Nash, M.D.*

DALE, W. ANDREW, and PEARSE, HERMAN E. Gas cysts of the intestines; report of two cases. *Surg., Gynec. & Obst.*, Feb., 1950, *90*, 215–220.

Gas cysts of the intestines (pneumatosis intestinalis, emphysematosis, pneumatosis cystoides intestinorum, etc.) occur rarely in humans although the condition has been recognized for years in hogs.

In general the syndrome consists of multiple thin-walled gas-containing cystic areas situated in the walls of the large or/and small intestines as well as in the areolar tissue of the mesentery and extraperitoneal areas. The cysts may be general in distribution or they may be localized. Occasionally, single air-bearing cysts have been found.

The condition is usually found at operation or at autopsy in conjunction with obstruction at the pyloric end of the stomach. The usual causes of pyloric obstruction are peptic ulcer or carcinoma. A bacterial and also a neoplastic cause for the formation of these cysts have been advanced but these etiologic factors seem improbable. It may be that gas is mechanically forced from the gastrointestinal tract into the bowel wall and adjacent tissues through the lymphatics. The strikingly similar lesion occurring in pigs has been produced experimentally by inducing a dietary deficiency in these animals. There may be some such association in humans since the patient with pyloric obstruction is usually malnourished.—*Mary Frances Vastine, M.D.*

BUSER, JULIAN W., KERSNER, JOSEPH B., and PALMER, WALTER L. Carcinoma of the large bowel, analysis of clinical features of 478 cases, including eighty five year survivors. *Cancer*, March, 1950, *3*, 214–228.

The experience of the University of Chicago Clinic and the Albert Merritt Billings Hospital

from 1930 to 1947 is the basis for this article. Of the 478 cases seen during this period, age, sex, and duration of symptoms are not of prognostic significance. Pain, occurring in 69.8 per cent of the patients, followed by rectal bleeding, weight loss and change in bowel habits were the commonest symptoms. The first symptom in lesions of the rectum and rectosigmoid was usually gross bleeding. This symptom decreased in frequency as distance from the anus increased. In contradistinction, pain was commonest in right-sided lesions and less frequent in distal portions of the colon.

The importance of roentgen examination has been well established. The correct positive diagnosis was made in 94 per cent of the cases. Fifteen carcinomas were undetected in the rectosigmoid and rectum. Only one was missed in the sigmoid. The superiority of digital and visual examination of the rectum is again emphasized.

Nearly half of the patients have metastasis before or at operation, but one-third of the patients who followed the advice of the physician may be expected to be alive and well five years later. The survival rate for carcinoma of the rectum is only one-half that of the remaining colon. The probability of recurrence seems to be much less after two years following the resection of the lesion.—*D. C. Gastineau, M.D.*

Lawson, Edwin H., and Whitener, Donald L. Retrograde jejunogastric intussusception; report of a case. *Arch. Surg.*, Feb., 1950, *60*, 242–246.

This article describes a case of retrograde intussusception at the site of an old posterior gastrojejunostomy, diagnosed at postmortem examination. There was no tumor present. Apparently no roentgenographic studies had been made.

The authors further review briefly the literature which included 46 cases of acute and 5 cases of subacute or chronic retrograde invagination. The diagnosis has been made in a few cases by fluoroscopic and roentgenographic demonstration of a movable filling defect in the stomach, with a uniform pattern of parallel curves suggestive of jejunal mucosa. Early surgery may result in 90 per cent recoveries. There is a 100 per cent mortality for the acute condition when surgical exploration is not attempted.—*P. R. Dirkse, M.D.*

Denneen, E. V., and Broderick, T. C. Gallstone intestinal obstruction. *Ann. Surg.*, Feb., 1950, *131*, 225–236.

The authors add 10 cases of gallstone intestinal obstruction with proved or presumptive cholecystoduodenal fistulas to the literature. The condition is uncommon but not rare, and among 12,153 cases of intestinal obstruction reported in 1943 gallstone was the cause in 208 instances.

The condition has occurred more frequently in females than males, and in persons over forty years of age when biliary lithiasis is more common. The gallstones usually enter the intestinal tract via a fistula between gallbladder and duodenum, or occasionally between gallbladder and colon. The sequence of events is cholelithiasis, acute cholecystitis, gangrene and perforation of the gallbladder against the duodenum and pressure necrosis of the calculus into the intestine. Stones smaller than 2.5 cm. may be passed without symptoms.

The majority of calculi lodge near the ileocecal region and produce the cardinal symptoms and signs of acute intestinal obstruction. The roentgenologist can be of great help by: (1) observing air or contrast medium in the biliary tract; (2) direct visualization or negative visualization of the gallstone; (3) change of position of a previously observed stone in the gallbladder; (4) evidence of complete or partial intestinal obstruction.

The treatment is immediate surgery after adequate preoperative management. The obstructing stone is removed, a search made for others, repair of the cholecystoduodenal fistula is contraindicated.—*G. P. Keefer, M.D.*

Kay, Saul, and Talbert, Pierre C. Adenoma of the liver, mixed type (hamartoma); report of two cases. *Cancer*, March, 1950, *3*, 307–315.

Two cases of hamartoma of the liver are reported by the authors. One patient was seven months old, the other thirteen years old. The tumors are rare, only 7 cases having been recorded. They consist of both proliferating hepatic cells and bile ducts, and are thus called "adenoma of the liver, mixed type." Histopathologically hamartoma resembles cirrhosis of the liver, but the authors feel that the two conditions are unrelated.—*J. G. Lorman, M.D.*

Radakovich, Michael. Epidermoid cyst of

the spleen; report of a case. *Ann. Surg.*, Feb., 1950, *131*, 268–276.

This case history of an epidermoid cyst of the spleen brings the total of this rare condition to 11 reported cases. Cysts of the spleen are rare and always present a difficult problem in diagnosis.

In this seventeen year old female, signs and symptoms were those of a large soft tissue mass in the left upper quadrant with slight left flank pain. Roentgen examination showed pressure displacement of the stomach, left kidney, small intestine, and splenic flexure of the colon associated with splenic enlargement of marked degree. Laparotomy with splenectomy and pathologic study of the specimen are necessary for accurate diagnosis.

Cysts of the spleen should be considered in the differential diagnosis of left upper quadrant tumor masses. A classification of splenic cysts is given in this report. The actual mode of origin of epidermoid cysts is open to speculation but an embryonal defect in the spleen is suggested by the specimen in this case.—*G. P. Keefer, M.D.*

GYNECOLOGY AND OBSTETRICS

Grünberger, V. Zur Klinik der mehrfachen primären Neoplasmen in der Gynäkologie. (Clinical feature of multiple primary neoplasms in gynecology.) *Wien. klin. Wchnschr.*, March 10, 1950, *62*, 173–178.

Grünberger reports 1,208 cases of multiple primary neoplasm that have been seen from 1936 to October, 1944, in the Gynecological Clinic of the University of Vienna.

The postulates of Billroth from the diagnosis of multiple primary cancers were used as the basis of this study: (1) Difference in histological structures. (2) Each one of the carcinomas has to be derived histogenetically from the epithelium of the original site. (3) Each of the carcinomas has to have its own metastases.

The report is concerned with 30 multiple primary carcinomas of which one was a triple carcinoma while the others were double growths. This gives a percentage of 2.5 per cent $\pm$0.4, a figure that is in agreement with the collected statistics of Warren and Gates. It seems that the occurrence and recognition of double carcinoma is rather favorable for an expectancy of cure as compared with single cancers. Eight of the 30 cases were reported as permanently cured, an absolute rate of 26.6 per cent which corresponds favorably with the percentage of the absolute cures of single primary carcinomas.—*Le Roy S. House, M.D.*

Tucker, Beatrice E., Solomkin, Mark, and Abrams, Bernard. A study of face presentation in the normal mature fetus. *Surg., Gynec. & Obst.*, Feb., 1950, *90*, 199–203.

From July 1, 1932, through September 30, 1948, 39,687 babies were delivered by the Chicago Maternity Center. Of this number, 73 were face presentations and of these 64 were "mature normal" babies. Six were born of primiparas and 58 of multiparas. Six were anencephalic monsters and 3 were premature babies.

Summary and Conclusions.

1. The etiology of face presentation is obscure. The only etiologic factors which could be substantiated by the authors' data were (1) multiparity and (2) original occiput posterior positions.

2. The presence of the cord around the neck did not differ materially from the incidence found in the service as a whole. However, fatal compression of the cord is more likely to occur in face presentation when the cord lies between the suboccipput and the back of the neck.

3. There was one prolapsed cord. This was associated with a major operative procedure and resulted in a neonatal death.

4. No significant difference was found in the weights of babies delivered by face and those via the usual cephalic presentations.

5. Face presentation showed a slightly shorter first stage of labor and a definitely longer second stage of labor than did the vertex presentation.

6. Most of the patients with face presentations will deliver spontaneously if permitted to do so.

7. In cases in which operative interference is indicated the simplest procedure will generally yield the most satisfactory results.

8. In general, the authors' low fetal mortality (for this position) was due, they believe, to a policy of non-interference while progress was being made.—*Mary Frances Vastine, M.D.*

SUBJECT INDEX TO VOLUME 64

(ab) = abstract (E) = editorial

Abdomen, pathophysiology of peculiar syndrome of acute distention of (ab), 689
preliminary roentgenogram of, in diagnosis of congenital obstruction of intestine, 430
prone position in roentgenological diagnosis of free intra-abdominal gas, 778
roentgen diagnosis of sponge in, 781
Abscess of liver, 785
pulmonary, as complication of hiatal hernia (ab), 688
unilateral tuberculous, of thoracic spine, with roentgen findings of dumb-bell neurofibroma (ab), 716
Achlorhydria, benign gastric ulcers occurring in presence of (ab), 159
Achondroplasia (ab) 346
Acne vulgaris, radiotherapy of (ab), 887
Acromegaly, study and treatment of (ab), 888
Acro-osteolysis, new clinical entity (ab), 553
Adenocarcinoma of body of uterus (ab), 891
of jejunum, massive resection of small bowel for (ab), 694
Adenocarcinomas, analogous mouse mammary, anomalous radiosensitivities of (ab), 557
Adenoleiomyomas, multiple, of both lungs (ab), 524
Adenoma, bronchial (ab), 153
fetal, of hypophysis, clinical and roentgen study (ab), 1026
of bronchus (ab), 153; (ab), 322
of liver, mixed type (ab), 1047
pituitary, roentgen therapy for (ab), 356
Adenomas, bronchial, clinical observation of (ab), 152
Adenomatosis, pulmonary alveolar (ab), 523; (ab), 679
Adenomyosis, gastric (ab), 331
Adenylic acid therapy, effect of, on pruritus due to Hodgkin's disease (ab), 896
Adrenal cortex, carcinoma of (ab), 702
Agenesis of corpus callosum (ab), 515
Agranulocytosis due to propylthiouracil (ab), 896
Albright's disease (ab), 176
Alimentary tract See Gastrointestinal tract
Allergy, gastrointestinal (ab), 331
American Association for Advancement of Science, announcement of meeting, 663
American College of Physicians, announcement of 1951 meeting, 307
research followships of, 306
American College of Radiology, 505
American Radium Society, announcement of 1951 meeting, 1021
program of 1950 meeting of, 305
American Roentgen Ray Society, Commercial Exhibit at 1950 meeting, 851
fiftieth anniversary of (E), 115
Fifty-first Annual Meeting of (E), 119; (E), 838
hotel reservations for Fifty-first Annual Meeting, 141
new officers for 1950–1951, 852
preliminary program of Fifty-first Annual Meeting, 126
Scientific Exhibit at 1950 meeting, 847
Section on Instruction for Fifty-first Annual Meeting, 130
Ampulla of Vater, premalignant lesions of (ab), 694
Amyloidosis (ab), 718
Anastomosis, end-to-end, of colon following resection, 769
Anatomy, roentgen, and interpretation of angiocardiograms (ab), 155
Anemia, familial erythroblastic, in American Indian infant (ab), 872
sickle cell, long bone involvement in (ab), 872
sickle-cell, roentgen findings in (ab), 716
Aneurysm, cerebral, relation of migraine to (ab), 1027
congenital, of superior vena cava (ab), 1040
dissecting, of aorta (ab), 1038
of great cerebral vein and circle of Willis (ab), 517
of pulmonary artery, 414
Aneurysms, arteriovenous, of brain (ab), 516
calcified cardiac (ab), 1041
multiple, of pulmonary arteries, roentgen diagnosis (ab), 533
Rasmussen's, and fatal hemorrhage, in pulmonary tuberculosis (ab), 320
Angiocardio-cinematography (ab), 904
Angiocardiograms, roentgen anatomy and interpretation of (ab), 155
Angiocardiography, 214; 255
demonstration of anomalous pulmonary circulation by, 409
diagnosis of coarctation of aorta by (ab), 1041
experimental, by direct puncture of heart (ab), 902
experimental study of effects of diodrast used in, 819
for investigation of thoracic tumors (ab), 323
full cycle (ab), 328
in thoracic surgery (ab), 686
problems of, 189
role of, in mediastinal and paramediastinal lesions (ab), 534
study of 53 consecutive proved cases of lung cancer by, 222
visualization of coronary circulation during (ab), 686
Angiocardiopneumography (ab), 684

Angiography, cerebral, complications following use of neo-iopax in, 816
cerebral (diodrast) transient hemiplegia associated with (ab), 515
cerebral, in brain tumor suspects (ab), 1027
cerebral, recorded cinefluorographically (ab), 1027
cerebral, review of (ab), 513
Angiomas, giant, treatment of (ab), 184
Angiomatosis, encephalotrigeminal (Sturge-Weber syndrome), 391
Angiosarcoma of colon (ab), 165
Ankle, footballer's (ab), 864
ligamentous fractures of, diagnosis and treatment (ab), 865
Anomaly, unusual, of superior extremities in Tarascan Indian girl (ab), 708
Anoxia, increased radioresistance of red bon marrow after (ab), 373
Anthracosilicosis, clinical evaluation of disability in (ab), 1032
Antritis, deforming, secondary to ulcer about incisura angularis of lesser curvature (ab), 543
Antrum, maxillary, epitheliomas of (ab), 357
prepyloric, fibromuscular atresia of (ab), 543
Anus, basal cell carcinoma of (ab), 361
Aorta, abdominal, congenital coarctation of (ab), 1041
Abdominal, roentgen study of, as aid to differential diagnosis between benign and malignant ulceration of stomach (ab), 332
abdominal, visualization of, and its branches following intravenous injection of contrast medium, 945
and great vessels, Valsalva maneuver in contrast visualization of (ab), 530
arch of, and its branches, congenital anomalies of (ab), 531
arteriography of, and branches, by polyethylene catheter, 196
ascending, calcification of, as diagnostic sign of syphilitic aortitis (ab), 532
coarctation of (ab) 532; (ab), 533
coarctation of, at level of diaphragm (ab), 534
diagnosis of coarctation of, by angiocardiography (ab), 1041
dilatation and pulsation of left subclavian artery in roentgen diagnosis of coarctation of (ab), 687
dissecting aneurysm of (ab), 1038
transposition of (ab), 531
vascularization of (ab), 534
Aortic bifurcation, thrombotic obliteration of (E), 496
Aortitis, syphilitic, calcification of ascending aorta as diagnostic sign of (ab), 532
Aortography, lumbar, Steno's experiment in man as complication in (ab), 717
Aplasia, medullary, therapeutic results of large sternosternal transfusions in (ab), 180
of lung, 409
of upper and middle lobe of right lung (ab), 153
Appendix, calculi of (ab), 334
Argentinian Radiological Meeting, 663
Arterial anomalies of spinal cord (ab), 341
Arteries, pulmonary, roentgen diagnosis of multiple aneurysms of (ab), 533
Arteriography, cerebral, 239; (ab), 1026
in cerebral arteriovenous malformation (ab), 675
of aorta and great vessels, Valsalva maneuver in technique of (ab), 530
of aorta and its branches by polyethylene catheter, 196
study by, of effect of drugs on intracranial vessels in patient with chronic headache (ab), 514
Arteriovenous malformation, cerebral, arteriography in (ab), 675
Artery, femoral, reconstruction of, for arteriosclerotic thrombosis (ab), 874
internal carotid, thrombosis of (ab), 313
left subclavian, dilatation and pulsation of, in roentgen diagnosis of coarctation of aorta (ab), 687
pulmonary, aneurysm of, 414
pulmonary, congenital anomalies causing enlargement of segment of, 214
Arthritis, gouty, diversity of, and its complications (ab), 175
psoriatic, 635
rheumatoid, and spondylitis, neutral 17-ketosteroids in (ab), 709
rheumatoid, effects of steroid compounds on various manifestations of (ab), 175
rheumatoid, general management of (ab), 708
rheumatoid, Morquio-Brailsford's disease simulating (ab), 709
Arthropathy, diabetic, and intercapillary glomerulosclerosis (ab), 711
Arthrosis, temporomandibular (ab), 342
Atelectasis, chronic, and pneumonitis of middle lobe (ab), 520
Atlanta Radiological Society, 1020
Atlanto-axial fracture-dislocation (ab), 174
joint, subluxation of (ab), 173
Atomic bomb, acute radiation injuries from (ab), 879
bomb radiation, radioresistant cells in certain radiosensitive tissues of swine exposed to (ab), 370
radiation, effects of, on oral and pharyngeal mucosa (ab), 721
Atresia, fibromuscular, of prepyloric antrum and its pathological explanation (ab), 543
Atrophy of heart (ab), 530
Atropine, pharmacodynamic test with, in gastroduodenal roentgen diagnosis (ab), 538

Back, low, anomalies of lumbosacral vertebrae in 550 individuals without symptoms referable to, 624

Backscatter, experimental study of, with x-ray beams of small area (ab), 188
Barium meal, very warm, study of normal and operated stomach with (ab), 543
Barotrauma, recurrent otitic, treatment of by irradiation (ab), 184
Beryllium, compounds in manufacture of radiotubes, diffuse pulmonary granulomatosis in young women following exposure to (ab), 524
skin granulomas from broken fluorescent tube (ab), 876
Besnier-Schaumann-Boeck syndrome (ab), 718
Beta ray burns of human skin (ab), 364
Betatron, depth dose of electrons from (ab), 722
Bile ducts, extra hepatic, carcinoma of (ab), 338
Biliary ducts, intrahepatic and extrahepatic, in aged, anatomo-roentgenographic studies in (ab), 551
lithiasis in young individuals (ab), 696
tract, gas-containing calculi of, 617
tract, roentgenologic and manometric exploration of, in surgery (ab), 551
Biological evaluation of 20 million volt roentgen rays, 963
Biophysical research work, roentgen apparatus for intense roentgen radiation of short duration intended for (ab), 889
Bladder, carcinoma, new technique for radium treatment of (ab), 359
cutaneous metastasis of carcinoma of (ab), 171
female urinary, cancer of (ab), 171
papilloma of (ab), 171
tumors of (ab), 172
Blastomycosis, acute primary pulmonary (ab), 1034
Blood from normal and leukemic subjects, effect of ultrasonic vibration on formed elements of (ab), 902
vascular origin, mediastinal tumors of (ab), 329
vessels, great, anatomo-roentgenological forms of transposition of (ab), 255
volume in heart and lungs and cardiac output through injection of radiophosphorus, determination of (ab), 363
Boeck's disease of lungs (ab), 680
Bone bank (ab), 710
cyst, aneurysmal (ab), 867
eosinophilic granuloma of (ab), 349
fibrous dysplasia of (ab), 349
formation, new multiple myeloma with (ab), 711
homogenous (bone bank), experiences in use of (ab), 174
homologous graft of preserved (ab), 710
irradiated, influence of energy absorption and electron range on dosage in (ab), 373
long, involvement in sickle cell anemia (ab), 872
marrow, red, increased radioresistance of, after anoxia (ab), 373
marrow studies in carcinoma of prostate (ab), 709
microradiography of (ab), 898
sclerosing conditions of (ab), 869
solitary plasmal cell myeloma of (ab), 868
tumors, diagnosis of (ab), 349
widespread metastases to, from carcinoma o-breast, radioactive phosphorus in treatment of, 559
Bones, long, fractures of (ab), 345
Bonoff, Karl M., 1892–1949, obituary, 501
portrait, 501
Book reviews
Roentgen-Ray Examination of the Digestive Tract. By Ross Golden, 144
Differential Diagnosis of Chest Diseases. By Jacob Jesse Singer, 144
Retroposition of Lumbar Vertebrae. By Lars Hagelstam, 144
Practical Neurological Diagnosis. By R. Glen Spurling, 145
Cancer. Hérédité—Hormones—Substances cancérigènes. By J. Maisin, 146
Clinical Radiation Therapy. Edited by Ernst A. Pohle, 146
Das menschliche Knochenmark. Seine Anatomie, Physiologie und Pathologie nach Ergebnissen der intravitalen Markpunktion. By Karl Rohr, 146
Röntgen- und Radiumphysik für Mediziner. By Richard Glocker, 147
Tabellen zur Dosierung der Röntgenstrahlen. By Leonhard Grebe and Werner Wiebe, 148
Manuel de Radiodiagnostic Clinique. By R. Ledoux-Lebard and Guy-R. Ledoux-Lebard, 506
Confrontations—Radio-Anatomo-Cliniques. Published under the direction of M. Chiray, R. A. Gutmann, and J. Sénèque, 506
Schilddrüse und Basedow. Beiträge zur Histo-Morphologie und Funktion der Schilddrüse verschiedener freilebender Tiere. By Wilhelm Eickhoff, 506
The Fundamentals of Pulmonary Tuberculosis and Its Complications. Edited by Edward W. Hayes, 507
Intestinal Intubation. By Meyer O. Cantor, 507
Bone and Joint Radiology. By Emerik Markovits, 508
Morgagni's Syndrome—Hyperostosis frontalis interna, Virilismus, Obesitas. By Folke Henschen, 509
Elementi di Anatomia Stratigrafica dello Scheletro. By Enzo Bullo and Roberto D'Alò, 509
Handbook of Materia Medica, Toxicology, and Pharmacology. By Forrest Ramon Davison, 510
Tomografía clínica cardio-vascular. By Juan Govea and Fidel Aguirre, 665

Die Durchleuchtungstechnik der Thoraxorgane. By E. A. Zimmer, 665
Radiologia Schermografica. By Franco Fossati and Patrizio Gallone, 665
An Atlas of Myelography. By Ernest H. Wood, Jr., 666
Urological Aspects of Spinal Cord Injuries. By George C. Prather, 666
Die Röntgendiagnostik der Wirbelsäule und ihre Grundlagen. By Adolf Liechti, 666
The 1949 Year Book of Radiology—(June, 1948—June, 1949). Edited by Fred Jenner Hodges, John Floyd Holt, and Isadore Lampe, 667
Aktuelle Probleme der Pathologie und Therapie. By H. Holthusen, 668
Myelography with Water-soluble Contrast, with Special Regard to the Normal Roentgenpicture. Sigfrid Arnell, 855
Congenital Anomalies of the Heart and Great Vessels. By Thomas J. Dry, Robert L. Parker, H. Milton Rogers, Jesse E. Edwards, Howard B. Burchell, and Arthur H. Bulbulian, 855
The Causation and Treatment of Delayed Union in Fractures of the Long Bone. By Kenneth W. Starr, 856
Carcinoma of the Larynx. By R. W. Gunderson, 856
Brookhaven Conference Report, 857
BRAIN, arteriovenous aneurysms of (ab), 516
metastatic melanoma of (ab), 1026
phantom, localization of radioactive materials in (ab), 554
tissue, tolerance dose of, to deep roentgen therapy (ab), 883
tumors, clinical use of radioactive phosphorus in surgery of (ab), 364
tumors of, complicating pregnancy (ab), 674
BREAST, cancer of (ab), 890
cancer, spontaneous regression of pleural carcinosis in (ab), 683
carcinoma, radioactive phosphorus in treatment of, 559
metastatic carcinoma of, lack of progressive growth of, transplanted subcutaneously into foreign strains of mice (ab), 901
BRILL-SYMMERS DISEASE (ab), 181
BRONCHI, adenoma of (ab), 153
carcinoma of (ab), 677
carcinoma, treatment of, with nitrogen mustard (ab), 894
clinical observation of benign tumors of (ab), 152
cytologic diagnosis of carcinoma of (ab), 678
deep roentgen therapy of cancer of, in association with progynon C treatment (ab), 886
inoperable carcinoma, nitrogen mustard in treatment of (ab), 895
major, tuberculosis of (ab), 321
obstruction of (ab), 677
BRONCHIAL secretions, cytologic study of cancer of lung by means of (ab), 522
tree, tuberculosis of (ab), 1036
BRONCHIECTASIS, pathogenesis of (ab), 528
study of (ab), 528
BRONCHIOLITHIASIS (ab), 1035
BRONCHITIS, tuberculous, in pulmonary resection (ab), 1034
BRONCHOGRAPHIC aspects of some pulmonary segments (ab), 317
BRONCHOGRAPHY, directed, with water soluble contrast media (ab), 528
in severely discharging lung (ab), 322
technique for instillation of lipiodol for (ab), 321
with iodized oil, lipoid granuloma of lung following (ab), 322
BRONCHUS, adenoma and cylindroma of (ab), 322
adenoma of (ab), 153
BROWN, PERCY, announcement of death of, 840

CAFFEY, JOHN, gives Maurice Lamm Blatt Memorial Fund Lecture, 854
CALCAREOUS deposits, multiple, in intestinal tract of newborn (ab), 546
CALCIFICATION, development of, in pulmonary lesions associated with sensitivity to histoplasmin (ab), 321
displaced intracranial physiologic, roentgen demonstration of (ab), 314
massive, in infarcted myocardium (ab), 529
of ascending aorta as diagnostic sign of syphilitic aortitis (ab), 532
of myocardium (ab), 155
pulmonary, tuberculosis? histoplasmosis?, 747
CALCIFICATIONS, cardiac valvular, roentgen kymographic study of (ab), 329
extensive metastatic, case of long standing renal insufficiency with, 423
of soft tissues associated with multiple destructive processes of phalanges of hand, 649
CALCINOSIS (ab), 874
universalis cutis, idiopathic, without disability (ab), 874
CALCIUM, acute deposition of, near elbow (ab), 863
CALCULI, appendical (ab), 334
gas-containing biliary 617
CALCULUS, urethral, in male (ab), 703
CALLUS, creeping etiology of (ab), 866
CALVÉ'S DISEASE, study of (ab), 351
CANCER, bronchial, deep roentgen therapy of, in association with progynon C treatment (ab), 886
early intestinal diagnosis of, 929
gastric, electrogastrography in diagnosis of (ab), 539
gastric, symptomatology and diagnosis of (ab), 1042
mammary, action of steroid hormones in (ab), 369

natural behavior of, in man (ab), 876
of breast (ab), 890
of breast, spontaneous regression of pleural carcinosis in (ab), 683
of cervix uteri, detection of (ab), 168
of cervix uteri, radiation therapy or surgery in (E), 299
of cervix uteri, radium needle therapy in, 1010
of colon in cases of colitis ulcerosa (ab), 549
of female urinary bladder (ab), 171
of lip (ab), 357
of lung, cytologic study of, by means of bronchial secretions (ab), 522
of lung, operability, 222
of lung, terminal bronchiolar or alveolar cell (ab), 324
of stomach, roentgen diagnosis of (ab), 690
of uterine cervix, treatment of rectovaginal fistulas following curie-roentgen therapy of (ab), 360
pelvic, planning of external irradiation in, 95
pelvic, planning of external irradiation in, correction, 1021
pelvic, radioactive colloidal gold in treatment of, 75
primary facial, combined radical cervicofacial procedures for (ab), 676
pulmonary, diagnosis and treatment of (ab), 521
research, British American exchange fellowships in, 853
research program, 141
results of treatment of (ab), 892
seminar, 306
thyroid, concentration of I^{131} in, shown by radioautography (ab), 363
uterine, new diagnostic adjunct for (ab), 169
Carcinoids of small intestine and stomach (ab), 549
Carcinoma, basal cell, of anus (ab), 361
bronchiogenic (ab), 677
bronchogenic, cytologic diagnosis of (ab), 678
bronchogenic, treatment of, with nitrogen mustard (ab), 894
cutaneous, treatment of, with podophyllin (ab), 368
early bronchogenic carcinoma, histological study of five minute pulmonary neoplasms believed to represent (ab), 324
early, of cervix uteri, diagnosis of (ab), 698
endometrial, genesis of (ab), 339
epidermoid, of penis (ab), 705
esophageal, combined roentgen-radium treatment of (ab), 885
esophageal, roentgen therapy of (ab), 358
hypopharyngeal, problem of (ab), 1024
inoperable bronchiogenic, treatment of, by nitrogen mustard (ab), 895
metastatic, involving central nervous system (ab), 706
metastatic mammary, lack of progressive growth of, transplanted subcutaneously into foreign strains of mice (ab), 901
of adrenal cortex (ab), 702
of bladder, cutaneous metastasis of (ab), 171
of bladder, new technique for radium treatment of (ab), 359
of breast, radioactive phosphorus in treatment of, 559
of cervix uteri, response of, to fractionated radium and roentgen therapy given concurrently, 446
of cervix uteri, results of treatment (ab), 892
of duodenum (ab), 163
of extrahepatic bile ducts (ab), 338
of large intestine (ab), 1046
of lip, results of treatment of (ab), 887
of lung, severe hypertrophic pulmonary osteoarthropathy due to, 42
of lung, study of 502 cases of (ab), 678
of prostate, bone marrow studies in (ab), 709
of prostate, myelofibrosis and generalized osteosclerosis secondary to (ab), 172
of stomach, extension of, into duodenum and esophagus (ab), 544
of thyroid gland (ab), 358
of thyroid of base of tongue (ab), 519
of uterine cervix complicating pregnancy (ab), 170
of vulva (ab), 359; (ab), 360
perforated, of stomach, simulating perforated gastric ulcer (ab), 544
primary, of duodenal bulb mimicking duodenal ulcer (ab), 163
primary, of infrapapillary portion of duodenum (ab), 1044
primary, of lung (ab), 1037
primary, of ureter (ab), 340; (ab), 703
primary, of vagina, radium therapy of, 86
pseudo-adenomatous basal cell, pulmonary metastases of (ab), 522
rectal, cauda equina syndrome due to (ab), 164
sclerosing, of stomach (ab), 162
squamous cell, of renal pelvis (ab), 170
visceral, and multiple venous thrombosis (ab), 550
Carcinosis, pleural, in breast cancer, spontaneous regression of (ab), 683
Cardia, gastric, diagnosis of lesions near (ab), 160
gastric, downward displacement of (ab), 1042
roentgen study of (ab), 331
Cardio-esophageal relaxation as cause of vomiting in infants (ab), 1030
Cardiology of old age (ab), 327
Cardiovascular anomalies, conventional roentgenography in diagnosis (ab), 328
effects of diodrast, experimental study of, 819
Carotid body, tumor of, associated with nodular goiter (ab), 676
tumors of (ab), 1028

Carpal bones, development of, as index of nutritional status of school children (ab), 713
tunnel compression of median nerves (ab), 343
Cartilage and bone, ectopic, experimental production of, in muscles of rabbits (ab), 174
Carty, John Russell, 1892–1949, obituary, 121
portrait, 121
Cast syndrome (E), 1013
Catheter, polyethylene, arteriography of aorta by means of, 196
Catheterization of left side of heart in man (ab), 1039
Cauda equina syndrome due to silent rectal carcinoma (ab), 164
Caustic substances, roentgen aspects of periesophageal changes in stenosis due to (ab), 330
Cavitary disease of lungs (ab), 1034
Cell nuclei, amphibian, nature of radiation injury to (ab), 373
Cells, radioresistant, in certain radiosensitive tissues of swine exposed to atomic bomb radiation (ab), 370
Cerebellum, metastasizing tumors of (ab), 517
Cerebral aneurysm, relation of migraine to (ab), 1027
angiography, complications following use of neo-iopax in, 816
angiography in brain tumor suspects (ab), 1027
angiography recorded cinefluorographically (ab) 1027
angiography, review of (ab), 513
arteriography, 239; (ab), 1026
Cervicofacial procedures, combined radical, for primary facial cancer (ab), 676
Cervix uteri, carcinoma of, complicating pregnancy (ab), 170
carcinoma, results of treatment (ab), 892
detection of cancer of (ab), 168
diagnosis of early carcinoma of (ab), 698
radiation therapy or surgery in cancer of (E), 299
radiation treatment of tuberculosis of (ab), 887
radium needle therapy in cancer of, 1010
response of carcinoma of, to fractionated radium and roentgen therapy given concurrently, 446
sarcoma of (ab), 361
treatment of rectovaginal fistulas following curie-roentgen therapy of cancer of (ab), 360
Chalasia as cause of vomiting in infants (ab), 1030
Charcot joints secondary to diabetes mellitus, 277
Chemical means, possibility of protecting living organism against roentgen rays by (ab), 904
Chemotherapy, experimental, of neoplastic diseases (ab), 367
of malignant tumors (ab), 369
of tumors, systemic pathological effects of nitrogen mustards in (ab), 365
Chest, angiocardiography in surgery of (ab), 686
lesions, fluoroscopic preselection of tomographic plane in (ab), 317
roentgen surveys of, in general hospitals (ab), 317
synovial sarcoma of wall of (ab), 1028
Chicago Roentgen Society, 142
Chick embryo, toxicity of roentgen rays to, 280
Chicken, toxicity and pathological effects of roentgen rays in, 289
Children, apparently normal six-year-old, summary of 49 radiologists' opinion on skeletal age limits of, 442
bronchopulmonary geotrichosis in (ab), 681
congenital obstructions of alimentary tract in (ab), 547
difficulties of differential diagnosis in tuberculosis of knee joint in (ab), 715
excisional surgical treatment of pulmonary tuberculosis in (ab), 680
gastric ulcer in, 765
initial roentgenographic signs of coxitis in (ab), 710
intervertebral lesions in (ab), 860
method for measuring hearts of (ab), 372
pneumonia in, following ingestion of petroleum products (ab), 525
radiology in neurosurgical conditions in (ab), 513
radon therapy of nasopharyngeal lymphoid hypertrophy in (ab), 181
small bowel obstruction in (ab), 545
school, carpal and epiphysial development as index of nutritional status of (ab), 713
spondylarthritis in, 20
stricture of urethra in (ab), 173
Cholecystocolic fistula (ab), 165
Cholecystography, intravenous, sodium tetraiodophthalicfluorescein for (ab), 166
new series of radiopaque compounds for (ab), 556
non-filling of gallbladder in (ab), 697
Cholelithiasis associated with peptic ulcer (ab), 551
Chondromas, primary, of lung (ab), 1033
Chondrosarcoma, primary, of lung (ab), 324
Cinefluorography, cerebral angiography recorded by (ab), 1027
Circle of Willis, arteriovenous fistula of (ab), 517
Circulation, anomalous pulmonary, angiocardiographic demonstration of, 409
coronary, visualization of, during angiocardiography (ab), 686
renal, in experimental hydronephrosis (ab), 906
Claudication, intermittent (ab), 180
Cleveland Radiological Society, 142
Coarctation, congenital, of abdominal aorta (ab), 1041
of aorta (ab), 532; (ab), 533
of aorta at level of diaphragm (ab), 534

of aorta, diagnosis of, by angiocardiography (ab), 1041
of aorta, dilatation and pulsation of left subclavian artery in roentgen diagnosis of (ab), 687
COCCIDIOIDOMYCOSIS, hazard involved in diagnostic procedures (ab), 720
residual cavities in pulmonary (ab), 720
studies on contagiousness of (ab), 901
COKE fires, pneumoconiosis after exposure to dust from (ab), 320
COLCHICIN, combination of, and roentgen therapy in leukemia (ab), 893
COLITIS ulcerosa, cancer of the colon in cases of (ab), 549
COLON, angiosarcoma of (ab), 165
cancer of, in cases of colitis ulcerosa (ab), 549
colonmetrographic studies of effects of section of parasympathetic nerves of (ab), 370
defunctioned, after ileostomy, roentgen examination of (ab), 164
descending, obstructive lesions of (ab), 165
distal, intestinal obstruction due to lesions of (ab), 550
diverticula of (ab), 336
end-to-end anastomosis of, following resection, 769
method for improving roentgenograms of, after colostomy, 835
perforation of, following use of enemas (ab), 337
polypoid lesions of (ab), 165
sigmoid, sigmoidovesical fistulas resulting from diverticulitis of (ab), 336
COLONMETROGRAPHIC studies of effects of section of parasympathetic nerves of colon (ab), 370
COLOSTOMY, method for improving roentgenograms of colon after, 835
CONES, precision, use of, in peroral irradiation, 968
CONGENITAL anomalies of aortic arch and its branches (ab), 531
cystic disease of lungs (ab), 319
heart disease, diagnosis of common types of (ab), 154
obstruction of stomach and small intestine in newborn (ab), 162
sternal fissure (ab), 316
CONTRAST media, experimental studies on, for use in neurography, 974
media, radiopaque, compounds of zirconium for (ab), 370
media, water soluble, directed bronchography with (ab), 528
medium (bismjol), results with use of, in retrograde pyelography (ab), 340
medium, visualization of abdominal aorta and its branches following intravenous injection of, 945
CORPUS CALLOSUM, agenesis of (ab), 515
roentgen diagnosis of lipoma of (ab), 516
CORTICAL inhibition of gastric motility (ab), 899
COXITIS in children, initial roentgenographic signs of (ab), 710
CRANIO-CLEIDO-DYSOSTOSIS (ab), 346
CRANIOPHARYNGIOMA in woman aged fifty-seven (ab), 673
CRANIOPHARYNGIOMAS, surgery of (ab), 518
CRETINISM, early detection of, by radioactive tracers (ab), 894
CYLINDROMA of bronchus (ab), 322
CYST, aneurysmal bone (ab), 867
calcified splenic (ab), 553
epidermoid, of spleen (ab), 1048
foregut, of mediastinum (ab), 681
large hydatid, of liver, 473
pancreatic (ab), 552
CYSTADENOCARCINOMA, papillary, of ovary, calcified metastatic deposits from, 467
CYSTIC disease, congenital, of lungs (ab), 319
CYSTOGRAPHIC studies in placenta praevia, 53
CYSTOSCOPY, evaluation of merits of, in management of renal trauma (ab), 699
CYSTOSTOMY, use of polythene and polyvinyl tubing in (ab), 341
CYSTOURETHROGRAPHY in diagnosis of diseases of prostatic urethra, 640
CYSTS, gas, of intestines (ab), 1046
hydatid, of liver, direct roentgen signs of (ab), 551
multiple duplicative, of duodenum causing acute intestinal obstruction (ab), 163
pylorenal (ab), 700
CYTOLOGY, exfoliative (ab), 352

DEAFNESS, nasopharyngeal radium applicator in treatment and prevention of (ab), 890
DETROIT ROENTGEN RAY AND RADIUM SOCIETY, 854
DIABETES MELLITUS, Charcot joints secondary to, 277
renal osteodystrophy associated with (ab), 701
DIAPHRAGM, fatal tension pneumothorax resulting from rupture of, in patient receiving pneumoperitoneum (ab), 526
right-sided stomach associated with eventration of, simulating hydropneumothorax, 50
temporary high position of (ab), 688
DIODRAST, experimental study of cardiovascular effects of, 819
transient hemiplegia associated with cerebral angiography using (ab), 515
Diphyllobothrium latum, roentgen visualization of, in intestine of man (ab), 155
DISEASE, systemic, concept and clinical reality of (ab), 353
DISLOCATIONS, inter-carpal, and fracture-dislocations (ab), 344
DIVERTICULA of alimentary tract (ab), 334; (ab), 335; (ab), 336
of colon (ab), 336
of esophagus (ab), 334
of stomach (ab), 159

of stomach and small intestine, roentgen aspects of (ab), 335
DIVERTICULITIS of large intestine (ab), 335
of sigmoid colon, sigmoidovesical fistulas resulting from (ab), 336
sigmoid, symptoms of obstruction from (ab), 164
DIVERTICULOSIS, intramural small-cystic, of gallbladder (ab), 696
of large intestine (ab), 335
DIVERTICULUM, Jeckel's, heterotopic pancreatic tissue and gastric mucosa in (ab), 696
DOSAGE, biologic, of roentgen rays (ab), 881
clinical radiation, interrelation of time, area and therapeutic ratio (ab), 371
in irradiated bone, influence of energy absorption and electron range on (ab), 373
DOSE calculator, improved, for linear radioactive sources (ab), 898
depth, of electrons from betatron (ab), 722
influence of fractionation of, on lethal roentgen-ray effect produced by total body irradiation in mice (ab), 556
tolerance, of brain tissue to deep roentgen therapy (ab), 883
units, tentative, for mixed radiations (ab), 906
DOSIMETER, calibrated roentgen-ray film badge, 475
DOSIMETRY of ionizing particles, 489
of radium containers (ab), 902
Drosophila, action of radioactive phosphorus in (ab), 362
bar effect in, in biological evaluation of 20 million volt roentgen rays, 963
DRUGS, arteriographic study of effect of, on intracranial vessels in patients with chronic headache (ab), 514
DUCTUS ARTERIOSUS, patent (ab), 682
DUODENUM, annular pancreas producing stenosis of (ab), 551
carcinoma of (ab), 163
etiology and pathogenesis of prolapsed gastric mucosa into (ab), 693
extrinsic obstruction of, in newborn (ab), 692
multiple duplicative cysts of, causing acute intestinal obstruction (ab), 163
preferential localization in, of case of lymphosarcomatous mycosis fungoides (ab), 1045
primary carcinoma of bulb of, mimicking duodenal ulcer (ab), 163
primary carcinoma of infrapapillary portion of (ab), 1044
prolapse of redundant gastric mucosa into (ab), 692
study of juxtapyloric portion of (ab), 1045
ulcers of, simulating prepyloric lesions, 603
DYSOSTOSES, polytopic hereditary enchondral (ab), 714
DYSPLASIA, cystic, of kidney (ab), 340
fibrous, of bone (ab), 349
multiple metaphyseal (ab), 708
progressive diaphyseal (ab), 713

EDEMA, pulmonary, and "batswing" shadow (ab), 521
pulmonary, experimental observations on dogs following acute peripheral blood loss (ab), 899
EDITORIALS
The Fiftieth Anniversary of the American Roentgen Ray Society, 115
The Annual Meeting of the American Roentgen Ray Society, 119
Arthur Fenwick Holding, 1877–1949, 120
John Russell Carty, 1892–1949, 121
Radiation therapy or surgery in cancer of the cervix uteri, 299
The Leriche syndrome (thrombotic obliteration of the aortic bifurcation), 496
George C. Johnston, 1872–1950, 499
Karl M. Bonoff, 1892–1949, 501
Sixth International Congress of Radiology, 655
The Fifty-first Annual Meeting of the American Roentgen Ray Society (Fiftieth Anniversary Meeting), 838
Robert Hervey Lafferty, 1878–1950, 841
Alfred Rosselet, 1887–1950, 842
The cast syndrome, 1013
ELBOW, acute deposition of calcium near (ab), 863
ELECTROGASTROGRAPHY in diagnosis of gastric cancer (ab), 539
ELECTROKYMOGRAPHY of heart and great vessels (ab), 326
present clinical status (ab), 327
ELECTRON accelerator, microwave linear (ab), 187
microscopy in relation to medical sciences (ab), 188
ELECTRONS, depth dose of, from betatron (ab), 722
EMBOLISM, fatal, following urethrography (ab), 701
EMPHYSEMA, bullous (ab), 318
fatal interstitial and mediastinal, following accidental needle perforations of trachea during jugular venipuncture (ab), 527
generalized subserous, through a single puncture roentgen diagnosis by, 723
mediastinal, 375
pulmonary, diagnosis (ab), 527
EMPHYSEMATOUS cavity, chronic, of lung (ab), 318
ENDOBRONCHIAL radium applicator (ab), 372
ENDOCRINE basis for slipping of upper femoral epiphysis (ab), 862
ENDOMETRIOSIS, pathogenesis of (ab), 682
ENDOMETRIUM, genesis of carcinoma of (ab), 339
ENEMAS, perforation of colon following use of (ab), 337
ENGELMANN'S DISEASE (ab), 713
ENTERITIS, chronic stenosing regional (ab), 333
regional, roentgen therapy of (ab), 886
EOSINOPHILIA, transitory, localized in knee joint after pneumarthrography (ab), 863

Epiphyses, destruction of, by frostbite (ab), 861
development of as index of nutritional status of school children (ab), 713
Epiphyses, upper femoral, endocrine basis for slipping of (ab), 862
upper femoral, slipping of (ab), 179
Epiphysitis, juvenile, possible relationship of osteitis condensans ilii to (ab), 869
Epitheliomas of maxillary antrum (ab), 357
Esophagectomy, transthoracic, gastropericardial fistula following (ab), 537
Esophagitis, nonspecific granulomatous (ab), 1029
Esophagus, benign tumors of (ab), 688
combined roentgen-radium treatment of carcinoma of (ab), 885
diaphragmatic, roentgen study of (ab), 331
diverticula of (ab), 334
gastric protrusions through hiatus of (ab), 1030
lower end of normal (ab), 535
rare tumors of (ab), 537
roentgen aspects in periesophageal changes in stenosis due to caustic substances (ab), 330
roentgen therapy of carcinoma of (ab), 358
short (ab), 536
spontaneous perforation of (ab), 536
spontaneous rupture of (ab), 536
Ethyl carbamate See Urethane
Extremities, gangrene of, of venous origin (ab), 873
superior, unusual anomaly of, in Tarascan Indian girl (ab), 708

Fascia, renal (ab), 702
Fellowships, British American Exchange, in cancer research, 853
Femur, aseptic necrosis of head of, simultaing tuberculosis (ab), 862
fracture of head of (ab), 179
intracapsular fracture of neck of (ab), 871
radioactive phosphorus in diagnosis of avascular necrosis of head of (ab), 893
upper epiphysis of, endocrine basis for slipping of (ab), 862
Fetus, detection of abnormalities of (ab), 168
face presentation in normal mature (ab), 1048
growth of biparietal diameter of, during last four weeks of pregnancy (ab), 707
tolerance of, to roentgen rays (ab), 888
Fibroma, giant intrathoracic (ab), 522
stenosing, of esophagus (ab), 537
Fibrosis, cystic, of pancreas, pulmonary disease associated with (ab), 1031
of pancreas (ab), 695
Fingers, roentgen therapy of osteomyelitis of, 810
Fissure, congenital sternal (ab), 316
Fistula, arteriovenous, of great cerebral vein and circle of Willis (ab), 517
cholecystocolic (ab), 165
gastropericardial, following transthoracic esophagectomy (ab), 537
Fistulas, rectovaginal, following curie-roentgen therapy of cancer of uterine cervix, treatment of (ab), 360
sigmoidovesical, resulting from diverticulitis of sigmoid colon (ab), 336
spontaneous renoduodenal (ab), 702
Florida Radiological Society, 854
Fluorescein as adjunct in treatment of radionecrotic ulcers (ab), 183
Fluorescent tube, broken, beryllium skin granulomas from (ab), 876
Footballer's ankle (ab), 864
Forearm, pronation injuries of (ab), 343
roentgen differentiation between torsion and rotation in fractures of (ab), 863
Forehead, tumors of (ab), 672
Fracture dislocation, atlanto-axial (ab), 174
dislocations (ab), 344
in animals with hypoparathyroidism treated with vitamin D_2, reparative osteogenesis of (ab), 557
intracapsular, of neck of femur (ab), 871
skull, mechanism of (ab), 1023
Fractures, effect of intramedullary nailing on healing of (ab), 347
fatigue, etiology of (ab), 866
ligamentous ankle, diagnosis and treatment (ab) 865
of dorsolumbar spine (ab), 177
of forearm, roentgen differentiation between torsion and rotation in (ab), 863
of long bones (ab), 345
of tibial plateau (ab), 864
Frostbite, epiphyseal destruction by (ab), 861
Functional disorders, roentgen therapy of (ab), 881

Gallbaldder, double (ab), 166; (ab), 338
intramural small-cystic diverticulosis of (ab), 696
non-filling of (ab), 697
Gallstone intestinal obstruction (ab), 1047
Gamma spectra emitted in transmutation of RaD →RaE (ab), 372
Gangrene of extremities of venous origin (ab), 873
Gas, free intra-abdominal, prone position in roentgenological diagnosis of, 778
in gastrointestinal tract (ab), 539
patterns, intestinal, as diagnostic aid, 959
pocket, isolated subhepatic, following punctiform perforation as complication of duodenal ulcer (ab), 540
Gas-containing biliary calculi, 617
Gases, toxic, pulmonary effects of (ab), 904
Gastritis, corrosive, clinical and experimental study of (ab), 157
giant hypertrophic (ab), 693
polypoid (ab), 1042
prepyloric local (ab), 332
Gastroduodenal roentgen diagnosis, pharmacodynamic test with atropine in (ab), 538
Gastrointestinal allergy (ab), 331

GASTROINTESTINAL TRACT See also Intestinal tract
bacterial synthesis of vitamin B_{12} in (ab), 879
congenital obstructions of, in infants and children (ab), 547
diverticula of (ab), 334; (ab), 335; (ab), 336
gas in (ab), 539
palpable tumors of, in region of small pelvis, roentgen visualization of (ab), 544
upper, combined medical and surgical management of hemorrhage of (ab), 541
GASTROSTOMY for malignant disease, evaluation of (ab), 690
GAUCHER'S DISEASE, adult (ab), 877
GEOTRICHOSIS, bronchopulmonary, in children (ab), 681
GLIOBLASTOMA multiforme, verified, study of 211 patients with (ab), 183
GLOMERULOSCLEROSIS, intercapillary, and diabetic arthropathy (ab), 711
GOITER, nodular, tumor of carotid body associated with (ab), 676
GOLD implants, automatic measuring apparatus for (ab), 555
radioactive colloidal metallic, use of, in treatment of malignant disease (ab), 720
GOUT, classical (ab), 866
GRAFT, homologous, of preserved bone (ab), 710
GRANULOMA, eosinophilic, of bone, pathological and clinical study of (ab), 349
lipoid, of lung following bronchography with iodized oil (ab), 322
GRANULOMAS, beryllium, from broken fluorescent tube (ab), 876
GRANULOMATOSIS, diffuse pulmonary, in young women following exposure to beryllium compounds in manufacture of radio tubes (ab), 524
GYNECOLOGY, multiple primary neoplasms in (ab), 1048

HALLUCINATIONS occurring in patients with cerebral lesions, genesis of (ab), 673
HAMARTOMA of liver (ab), 1047
of lung (ab), 1033
HAND, multiple destructive processes of phalanges of, with simultaneous calcifications of soft tissues, 649
HARRIS tube, intestinal intubation with (ab), 540
HEAD trauma, roentgenologic aspects of, 399
HEADACHE, chronic, arteriographic study of effect of drugs on intracranial vessels in patients with (ab), 514
HEART, and great vessels, electrokymography of (ab), 326
apparatus and technique for demonstration of chambers of, by roentgen cinematography (ab), 904
atrophy of (ab), 530
calcified aneurysms of (ab), 1041
catheterization of left side of, in man (ab), 1039
complications of, in rheumatic fever (ab), 1040
configurations due to valvular disease, roentgen diagnosis of (ab), 155
determination of blood volume in, through injection of radiophosphorus (ab), 363
development of, in left-sided position (ab), 687
dilatation of, and exudative pericarditis, differential diagnosis (ab), 531
disease, congenital, diagnosis of common types of (ab), 154
disease, congenital, roentgen cinematography in (ab), 530
disease, congenital, studies of (ab), 1041
disease, operable congenital, conventional roentgen examination in (ab), 328
drainage of pulmonary veins into right side of (ab), 326
experimental angiocardiography by direct puncture of (ab), 902
in children, method for measuring (ab), 372
roentgen kymographic study of valvular calcifications of (ab), 329
study of enlargement of, in infancy (ab), 687
HEMANGIOBLASTOMAS, occurrence of, in three members of a family (ab), 341
HEMANGIOMA, cavernous, of lung (ab), 1033
of knee joint (ab), 178
HEMANGIOMAS, cavernous, radiation therapy of (ab), 883
radioresistant (ab), 185
HEMANGIOPERICYTOMA (ab), 717
HEMANGIO-SARCOMATOSIS, generalized, erroneously considered as generalized tuberculosis (ab), 1034
HEMATOMA, subdural, and effusion in infants (ab), 313
HEMATOMAS, intracerebral, roentgenographic visualization of, following prefrontal lobotomy, 47
HEMIPLEGIA, transient, associated with cerebral angiography (diodrast) (ab), 515
HEMOPATHY, malignant, treated with nitrogen mustard (ab), 368
HEMORRHAGE of upper gastrointestinal tract, combined medical and surgical management of (ab), 541
HERNIA, diaphragmatic, with complete evisceration of liver (ab), 329
hiatal, pulmonary abscess as complication of (ab), 688
intra-abdominal, case of, with incarceration of jejunum into transverse mesocolon, 940
HERNIATION of intervertebral discs, contralateral symptoms and signs produced by (ab), 760
HERPES ZOSTER after irradiation (ab), 355
HIDRADENITIS suppurativa axillaris, roentgen treatment of (ab), 885
HIP, posterior dislocation of (ab), 179
roentgen examination of, in Legg-Perthes' disease (ab), 347

Hirschsprung's disease (ab), 336
Histochemography, preliminary report on (ab), 368
Histoplasmin, development of calcification in pulmonary lesions associated with sensitivity to (ab), 321
Histoplasmosis, pulmonary calcification in, 747
roentgen semeiology of (ab), 719
Hodgkin's disease, effect of adenylic acid therapy on pruritus due to (ab), 896
long remissions in (ab), 873
management of, 913
medullary compression by (ab), 707
of lung, roentgen appearance and therapy of (ab), 1033
treatment of, with roentgen irradiation and nitrogen mustard, 61
Holding, Arthur Fenwick, 1877–1949, obituary, 120
portrait, 120
Hormones, steroid, action of, in mammary cancer (ab), 369
Hospitals, general, chest roentgen-ray surveys in (ab), 317
Hydrocalycosis (ab), 170
Hydronephrosis, contribution to study of (ab), 557
experimental, renal circulation in (ab), 906
Hydropneumothorax, right-sided stomach associated with eventration of diaphragm simulating, 50
Hydroxyphenyl fatty acids, iodinated, cholecystographic uses of (ab), 556
Hyperostoses, infantile cortical (ab), 869
Hyperparathyroidism (ab), 175
Hypertrophy, nasopharyngeal lymphoid, radon therapy of, in children (ab), 181
pyloric, in adult (ab), 1044
Hyperthyroidism, I^{131} in diagnosis and treatment of, 576
Hypoparathyroidism, psuedo (ab), 875
reparative osteogenesis of fracture in animals with, treated with vitamin D_2 (ab), 557
Hypopharyngeal region, problem of carcinoma of (ab), 1024
Hypophyseal region, diencephalic, functional irradiation of (ab), 884
Hypophysis, fetal adenoma of (ab), 1026

I^{131}, concentration of, in thyroid cancer, shown by radioautography (ab), 363
in diagnosis and treatment of hyperthyroidism, 576
Ileitis, regional, recurrence in (ab), 549
Ileocecal valve, normal, roentgen-anatomical studies of (ab), 695
Indiana Roentgen Society, 1021
Infant, American Indian, familial erythroblastic anemia in (ab), 872
malignant melanoma in (ab), 877
newborn, diagnosis of surgical conditions of (ab), 875
Infants, cardio-esophageal relaxation as cause of vomiting in (ab), 1030
congenital obstructions of alimentary tract in (ab), 547
genetic connection between pyloric hypertrophy in, and in adults (ab), 691
hypertrophic pyloric stenosis in, roentgen examination in (ab), 546
small bowel obstruction in (ab), 545
study of cardiac enlargement in (ab), 687
subdural hematoma and effusion in (ab), 313
table unit for fluoroscopic examination of (ab), 1028
Inflammatory lesions of small intestine, roentgen diagnosis of (ab), 1045
International Congress of Cancer, Fifth, 141
International Congress of Radiology, Sixth (E), 655
Intervertebral disc herniation, air and pantopaque myelograms in diagnosis of sciatica from (ab), 342
disc lesions in children and adolescents (ab), 860
discs, herniated, producing contralateral symptoms and signs (ab), 860
Intestinal tract. See also Gastrointestinal tract
of newborn, multiple calcareous deposits in (ab), 546
Intestine, diagnosis of early cancer of, 929
gas patterns in, as diagnostic aid, 959
large, carcinoma of (ab), 1046
large, diverticulosis and diverticulitis of (ab), 335
multiple duplicative cysts of duodenum causing acute obstruction of (ab), 163
obstruction of, due to lesions of distal colon (ab), 550
obstruction of, in newborn (ab), 545
of man, roentgen visualization of fish tapeworm, *Diphyllobothrium latum* in (ab), 155
preliminary roentgenogram of abdomen in diagnosis of congenital obstruction of, 430
physics in intubation of (ab), 694
small, carcinoids of (ab), 549
small, diagnosis of congenital obstruction of, in newborn (ab), 162
small, massive resection of, for adenocarcinoma of jejunum (ab), 694
small, obstruction of, in infancy and childhood (ab), 545
small, roentgen aspects of diverticula of (ab), 335
small, roentgen diagnosis of inflammatory lesions of (ab), 1045
small, variability of mucosa of (ab), 548
Whipple's lipodystrophy of (ab), 547
Intestines, gas cysts of (ab), 1046
roentgen aspects of occlusions of (ab), 333
Intracavitary therapy, new type of roentgen periscope for, 492
Intravasation, venous, during myelography (ab), 707

Intubation, intestinal, with Harris tube, technique and results (ab), 540
intestinal, physics in (ab), 694
Intussusception, retrograde jejunogastric (ab), 1047
Iodine, radioactive, distribution of, in mice with and without tumor 15091a after injection of radioactive sodium iodide (ab), 905
Iodized oil, lipoid granuloma of lung following bronchography with (ab), 322
Ionization methods, experimental determination by, of rate of emission of beta- and gamma-ray energy by radioactive substances (ab), 186
Ionizing particles, dosimetry of, 489
Irradiation, differentiation and (ab), 881
herpes zoster after (ab), 355
peroral, use of precision cones in, 968
resistance of Sciara to mutagenic effects of (ab), 371
total body, in mice, influence of dose fractionation on lethal roentgen-ray effect produced by (ab), 556
Ischiopubic junction, juvenile, transformation zones of (ab), 867
Isodose curves, clinical, 999
measurements of linear radium sources in air and water by means of automatic isodose recorder, 989

Jejunum, massive resection of small bowel for adenocarcinoma of (ab), 694
Johnston, George C., 1872–1950, obituary, 499
portrait, 499

Kidney, cystic dysplasia of (ab), 340
effect of tetra-ethyl ammonium bromide on pelvis of (ab), 557
lethargy of (ab), 699
Kidneys, circulation of, in experimental hydronephrosis (ab), 906
fascia of (ab), 702
nephrolithiasis with tubular failure of (ab), 700
squamous cell carcinoma of renal pelvis (ab), 170
trauma of, cystoscopy and retrograde pyelography in management of (ab), 699
Knee, pneumoarthrography of, as diagnostic aid (ab), 711
Knee joint, hemangioma of (ab), 178
transitory eosinophilia localized in, after pneumarthrography (ab), 863
tuberculosis of, in children, difficulties of differential diagnosis (ab), 715
Kymography, roentgen study of cardiac valvular calcifications by (ab), 329
roentgen, study of coarctation of aorta by (ab), 687

Lacteal duct, papilloma of (ab), 519
Lafferty, Robert Hervey, 1878–1950, obituary, 841
portrait, 841
Legg-Perthes' disease, roentgen examination of hip in (ab), 347
Leriche syndrome (E), 496
Lesions, premalignant, of ampulla of Vater (ab), 694
Leukemia, acute, therapeutic results of large sternosternal transfusions in (ab), 180
aleukemic myelogenous, diffuse osteosclerosis in (ab), 867
combination of colchicin and roentgen therapy in (ab), 893
diagnosis and treatment of, from standpoint of radiologist, 919
effect of ultrasonic vibration on formed elements of blood in (ab), 902
present status of radiation therapy (ab), 184
treatment of, 907
Levocardia, study of (ab), 687
Linitis plastica (ab), 162
Lip, cancer of (ab), 357
carcinoma, results of treatment of (ab), 887
Lipodystrophy, Whipple's intestinal (ab), 547
Lipoma of corpus callosum, roentgen diagnosis of (ab), 516
submucosa ventriculi simulating cancer of stomach (ab), 161
submucosal gastric, interesting roentgen sign in, 938
Lithiasis, biliary, in young individuals (ab), 696
Liver, abscess of, 785
diaphragmatic hernia with complete eviscera-tion of (ab), 329
direct roentgen signs of hydatid cysts of (ab), 551
large hydatid cyst of, 473
mixed type adenoma of (ab), 1047
Lobotomy, prefrontal, roentgenographic visualization of intracerebral hematomas following, 47
Localization, in vivo, of radioactive silver at predetermined sites in tissues, 831
Loeffler's syndrome, occurrence in two patients treated with penicillin in oil and wax (ab), 524
Looser-Milkman's syndrome with neurofibromatosis Recklinghausen and general decalcification of skeleton (ab), 714
Looser's transformation zones (ab), 867
zones, etiology of (ab), 866
Lumbosacral abnormalities, congenital, significance of (ab), 350
Lunatomalacia (ab), 345
Lung, aplasia of, 409
cancer of, terminal bronchiolar or alveolar cell (ab), 324
chronic atelectasis and pneumonitis of middle lobe of (ab), 520
chronic emphysematous cavity of (ab), 318

edema of, and "batswing" shadow (ab), 521
lipoid granuloma of, following bronchography with iodized oil (ab), 322
right, aplasia of upper and middle lobe of (ab), 153
severely discharging, bronchography in (ab), 322
wounded, delayed pathology of (ab), 325
Lungs, abscess of, as complication of hiatal hernia (ab), 688
acute primary blastomycosis of (ab), 1034
alveolar adenomatosis of (ab), 679
alveolar cell tumors of, 1
Boeck's disease of (ab), 680
both, multiple adenoleiomyomas of (ab), 524
bronchographic aspects of segments of (ab), 317
calcification of, tuberculosis? histoplasmosis?, 747
cancer, cytologic study of, by means of bronchial secretions (ab), 522
carcinoma of, 42
carcinoma, study of 502 cases (ab), 678
cavernous hemangioma of (ab), 1033
cavitary disease of (ab), 1034
changes in, in the reticuloses (ab), 1038
congenital cystic disease of (ab), 319
development of calcification in lesions of, associated with sensitivity to histoplasmin (ab), 321
diagnosis and treatment of cancer of (ab), 521
diagnosis of emphysema of (ab), 527
different behavior of different segments of (ab), 676
diffuse granulomatosis of, in young women following exposure to beryllium compounds in manufacture of radio tubes (ab), 524
disease of, associated with cystic fibrosis of pancreas (ab), 1031
edema of, experimental observations on dogs following acute peripheral blood loss (ab), 899
effects on, of toxic gases and smokes (ab), 904
hamartoma of (ab), 1033
Hodgkin's disease of, roentgen appearance and therapy (ab), 1033
manifestations of scleroderma in (ab), 679
net-like appearance of apex of (ab), 318
operability of cancer of, 222
primary carcinoma of (ab), 1037
primary chondromas of (ab), 1033
primary chondrosarcoma of (ab), 324
re-expansion of, in artificial pneumothorax of (ab), 1037
studies on respiratory gas exchange in anthracite coal miners with affections of (ab), 1031
Lymphoblastoma, diagnosis and treatment of, from standpoint of radiologist, 919
gross follicular (ab), 181
Lymphogranulomatosis benigna pulmonum (ab), 680
Lymphomas, treatment of, with nitrogen mustard (ab), 894
Lymphosarcoma of prostate (ab), 172

Maine Radiological Society, 505
Malignant disease, evaluation of gastrostomy for (ab), 690
use of radioactive colloidal metallic gold in treatment of (ab), 720
Marrow inhibition, control of polycythemia by (ab), 361
Maurice Lamm Blatt Memorial Fund Lecture, 854
Maxillary sinusitis, chronic, pathology, diagnosis and treatment of (ab), 316
Measurements, isodose, of linear radium sources in air and water by means of automatic isodose recorder, 989
Mecholyl, production of gastric and duodenal ulcers by prolonged administration of (ab), 187
Meconium peritonitis (ab), 156
Mediastinal emphysema, 375
Mediastinum, foregut cyst of (ab), 681
role of angiocardiography and venography in lesions of (ab), 534
tumors of, of blood vascular origin (ab), 329
Medical school, radiology in, 795
sciences, electron microscopy in relation to (ab), 188
Melanoma, malignant, in infant (ab), 877
metastatic, of brain (ab), 1026
Melorheostosis Léri, 789
Meningioma, hyperostosing, of pterion (ab), 1025
intracranial, following trauma (ab), 675
Meningiomas of sphenoidal ridge, roentgen diagnosis of (ab), 314
Meniscus, torn discoid (ab), 871
Mesenteric thrombosis, roentgen examination in, 610
Metacarpal bone, first, congenital abnormality of (ab), 344
Metaphyseal dysplasia, multiple (ab), 708
Metastases, calcified, from papillary cystadenocarcinoma of ovary, 467
pulmonary, of pseudo-adenomatous basal cell carcinoma (ab), 522
to bone from carcinoma of breast, radioactive phosphorus in treatment of, 559
Metastasis, cutaneous, of bladder carcinoma (ab), 171
Metastatic calcifications, case of long standing renal insufficiency with, 423
Metatarsalgia, Morton's (ab), 343
Metopic suture, roentgen study of (ab), 672
Microradiography of bone (ab), 898
Migraine, relation of, to cerebral aneurysm (ab), 1027
Miners, anthracite coal, with pulmonary complaints studies on respiratory gas exchange in (ab), 1031

Minnesota Radiological Society, 307
Mononucleosis, infectious, roentgenological aspect of, 950
Monster, double, roentgen diagnosis of (ab), 698
Morquio-Brailsford's disease simulating arthritic manifestation of rheumatoid disease (ab), 709
Mouth, use of precision cones in irradiation of lesions about, 968
Mucosa, gastric, prolapse of, through pylorus, roentgen aspect (ab), 332
of small bowel, variability of (ab), 548
oral and pharyngeal, effects of atomic radiation on (ab), 721
prolapsed gastric, into duodenum, etiology and pathogenesis of (ab), 693
redundant gastric, prolapse of, into duodenum (ab), 692
Muscles of rabbits, experimental production of ectopic cartilage and bone in (ab), 174
Myasthenia gravis, symptoms of, in cystic tumor of thymic region (ab), 1029
Mycosis, bronchopulmonary (ab), 1035
fungoides, ethyl carbamate in treatment of (ab), 897
fungoides, lymphosarcomatous, preferential localization in duodenum of (ab), 1045
Myelofibrosis secondary of carcinoma of prostate (ab), 172
Myelograms, air and pantopaque, in diagnosis of sciatica from disc herniation (ab), 342
Myelography, pantopaque (ab), 861
venous intravasation during (ab), 707
Myeloma, multiple, effect of urethane therapy on, 799
multiple, treatment of, with urethane (ab), 896
multiple, variability of roentgen appearance, 799
multiple, with new bone formation (ab), 711
solitary plasma cell, of bone (ab), 868
Myocardium, calcification of (ab), 155
infarcted, massive calcification in (ab), 529
Myositis ossificans progressiva (ab), 878
Myxedema, pituitary (ab), 674

Nasopharyngeal tumors (ab), 315
Neck, neurofibrosarcoma of, case with ten year survival after roentgen therapy, 470
Necrosis, acute, of renal papillae in pyelonephritis (ab), 702
aseptic, of femoral head simulating tuberculosis (ab), 862
avascular, of femoral head, radioactive phosphorus in diagnosis of (ab), 893
Neo-iopax, complications following use of, in cerebral angiography, 816
Neoplasm, pulmonary, cavitary form of (ab), 152
Neoplasms, five minute pulmonary, believed to represent early bronchogenic carcinoma, histological study of (ab), 324
multiple primary, in gynecology (ab), 1048
ulcerated, of stomach, roentgen findings in (ab), 690
Neoplastic diseases, clinical effect of nitrogen mustard on (ab), 366
experimental chemotherapy of (ab), 367
Nephrography, experimental and clinical observations (ab), 340
Nephrolithiasis with renal tubular failure (ab), 700
Nephrostomy, use of polythene and polyvinyl tubing in (ab), 341
Nerves, parasympathetic, of colon, colonmetrographic studies of effects of section of (ab), 370
Nervous system, central, metastatic carcinoma involving (ab), 706
Neurofibroma, solitary intrathoracic (ab), 325
Neurofibromatosis, multiple, sarcomatous transformation in (ab), 879
Neurofibrosarcoma of neck, case with ten year survival after roentgen therapy, 470
Neurography, experimental, 974
Neuromas, acoustic, roentgenologic manifestations of, 265
Neurosurgery, roentgenological aspects before and after, in encephalotrigeminal angiomatosis, 391
Neurosurgical conditions in childhood, radiology in (ab), 513
Newborn, diagnosis of congenital obstruction of stomach and small intestine in (ab), 162
extrinsic duodenal obstruction in (ab), 692
intestinal obstruction in (ab), 545
multiple calcareous deposits in intestinal tract of (ab), 546
New England Roentgen Ray Society, 663
New York Roentgen Society, 143
Nitrogen mustard, clinical effect of, on neoplastic diseases (ab), 366
effects of, on regenerating rat liver (ab), 365
in treatment of inoperable bronchiogenic carcinoma (ab), 895
malignant hemopathy treated by (ab), 368
statistical analysis of effects on sarcoma 180 and viscera of normal mice (ab), 366
systemic pathological effects of, in chemotherapy of tumors (ab), 365
testicular lesions in mice following parenteral administration of (ab), 365
treatment of bronchogenic carcinoma by (ab), 894
treatment of Hodgkin's disease with, 61; 913
treatment of lymphomas and other neoplastic diseases with (ab), 894

Oak Ridge summer symposium, 142
Obstruction, bronchial (ab), 677
congenital, of intestine, preliminary roentgenogram of abdomen in diagnosis of, 430
extrinsic duodenal, in newborn (ab), 692

gallstone intestinal (ab), 1047
intestinal, due to lesions of distal colon (ab), 550
intestinal, in newborn (ab), 545
small bowel, in infancy and childhood (ab), 545
Obstructions, congenital, of alimentary tract in infants and children (ab), 547
Occlusion, mesenteric vascular (ab), 550
Oklahoma State Radiological Society, 307
Old age, cardiology of (ab), 327
Oregon Radiological Society, 307
Organism, living, possibility of protecting, against roentgen rays by chemical means (ab), 904
Osteitis condensans ilii, relationship to juvenile epiphysitis (ab), 869
deformans, osteogenic sarcoma as complication of (ab), 175
fibrosa cystica, renal, 423
pubis (ab), 348
Osteoarthritis of trapezio-metacarpal joint (ab), 344
Osteoarthropathy, severe hypertrophic pulmonary, 42
Osteochondromatosis, synovial (ab), 178; (ab), 348
Osteodystrophy, renal associated with diabetes mellitus (ab), 701
Osteogenesis, reparative, of fracture in animals with hypoparathyroidism treated with vitamin D_2 (ab), 557
Osteoid osteoma (ab), 866
Osteomalacia (ab), 861
Osteomas of paranasal sinuses, treatment (ab), 671
Osteomyelitis of fingers, roentgen therapy of, 810
planar roentgenography in (ab), 898
sickle cell disease simulating (ab), 872
Osteopathia striata (ab), 870
Osteoporosis, roentgen appearance of, in Theresienstadt concentration camp (ab), 349
Osteosclerosis, diffuse, in aleukemic myelogenous leukemia(ab), 867
generalized, secondary to carcinoma of prostate (ab), 172
Ovary, calcified metastatic deposits from papillary cystadenocarcinoma of, 467

Pain, relief of, by roentgen therapy (ab), 888
roentgen therapy of (ab), 356
Pancreas, annular, producing duodenal stenosis (ab), 551
cyst of (ab), 552
fibrosis of (ab), 695
pulmonary disease associated with cystic fibrosis of (ab), 1031
Pancreatitis, calcareous (ab), 166
Papilloma of bladder (ab), 171
of lacteal duct (ab), 519
Paraffin pneumonia (ab), 525
Parathyroid gland, malignant tumors of (ab), 1029
Pelvimetry, orthographic (ab), 167
roentgen, method of, 505
Pelvis, planning of external irradiation in cancer of, 95
radioactive colloidal gold in treatment of cancer of, 75
use of roentgenograms in assessing disproportion of (ab), 553
Penicillin in oil and wax, occurrence of Loeffler's syndrome in two patients treated with (ab), 524
Penis, epidermoid carcinoma of (ab), 705
Peptic ulcer See also Ulcer
about incisura angularis of lesser curvature, deforming antritis secondary to (ab), 543
benign, occurring in presence of achlorhydria (ab), 159
cholelithiasis associated with (ab), 551
evidence relating life situation and emotional response to (ab), 159
findings in roentgen examination of 1,700 cases of, during and after World War II (ab), 161
following splanchnicectomy (ab), 542
isolated subhepatic gas pocket following punctiform perforation as complication of (ab), 540
limitations of vagotomy in treatment of (ab), 542
perforated, perforated carcinoma of stomach simulating (ab), 544
primary carcinoma of duodenal bulb mimicking (ab), 163
production of, by prolonged administration of mecholyl (ab), 187
recurrence of, after complete vagotomy (ab), 542
roentgen appearance of type of, described as dome shaped (ab), 333
roentgenological and surgical reports in, of duodenal bulb (ab), 541
roentgenoscopy in diagnosis of (ab), 156
Pericarditis, exudative, and cardiac dilatation, differential diagnosis (ab), 531
Peritonitis, meconium (ab), 156
Pes cavus (ab), 865
Petroleum products, pneumonia in children following ingestion of (ab), 525
Phalanges of hand, multiple destructive processes of, with simultaneous calcifications of soft tissues, 649
Pharmacodynamic test with atropine in gastroduodenal roentgen diagnosis (ab), 538
Pheochromocytoma, intrathoracic (ab), 681
intrathoracic, with hypertension (ab), 523
Phlebography, results of, and radical operative treatment, in post-thrombotic varices (ab), 179
Phosphorus, radioactive See also Radiophosphorus
radioactive, action of, in *Drosophila* (ab), 362
radioactive, clinical use of, in surgery of brain tumors (ab), 364

radioactive, in diagnosis of avascular necrosis of femoral head (ab), 893
radioactive, production of malignant tumors in rats with (ab), 905
radioactive, use of, in treatment of carcinoma of breast with widespread metastases to bone, 559
Photofluorographic methods, attempts at gastric examination by (ab), 897
Phototimer, improved roentgen-ray (ab), 898
Pineal tumors, metastatic (ab), 1025
Pittsburgh Roentgen Society, 663
Pituitary myxedema (ab), 674
Placenta praevia, cystographic studies in, 53
praevia, roentgen diagnosis of (ab), 339
roentgen localization of site of (ab), 339
Placental site, roentgen localization of (ab), 167
Plasmocytoma, solitary, of spine (ab), 177
Pleonosteosis, Léri's (ab), 343
Pleural effusions, observations on planar roentgenographic examination in (ab), 318
Pneumoarthrography of knee as diagnostic aid (ab), 711
transitory eosinophilia localized in knee joint after (ab), 863
Pneumoconiosis after exposure to sulphur dioxide fumes and dust from coke fires (ab), 320
Pneumoencephalography, subdural fluid as consequence of (ab), 1025
Pneumonia, diagnosis of, preceding tuberculosis (ab), 525
in children following ingestion of petroleum products (ab), 525
paraffin (ab), 525
Pneumonitis, and chronic atelectasis of middle lobe (ab), 520
chronic (ab), 319
diffuse miliary granulomatous, of unknown etiology, roentgen aspects of, 740
Pneumopericardium, disease process of (ab), 1039
Pneumoperitoneum, fatal tension pneumothorax resulting from diaphragmatic rupture in patient receiving (ab), 526
Pneumothorax cavity, extrapleural, free coagula within (ab), 529
chronic spontaneous, surgical management of (ab), 1037
extrapleural, roentgen aspects of (ab), 688
fatal tension, resulting from diaphragmatic rupture in patient receiving pneumoperitoneum (ab), 526
lung, artificial, re-expansion of (ab), 1037
recurrent and chronic spontaneous, of nontuberculous origin, surgical treatment of (ab), 526
Podophyllin, treatment of cutaneous carcinoma with (ab), 368
Polycythemia, control of, by marrow inhibition (ab), 361
treatment of, 907
Polypoid lesions of colon and rectum (ab), 165
Polythene and polyvinyl tubing, use of, in ureterostomy, nephrostomy and cystostomy (ab), 341
Pregnancy, carcinoma of uterine cervix complicating (ab), 170
growth of fetal biparietal diameter during last four weeks of (ab), 707
secondary abdominal (ab), 698
so-called angular (ab), 697
tumors of brain complicating (ab), 674
Premalignant lesions of ampulla of Vater (ab), 694
Presentation, face, in normal mature fetus (ab), 1048
Proctitis, factitial (irradiation) (ab), 181
Progynon C treatment of bronchial cancer in association with deep roentgen therapy (ab), 886
Propylthiouracil, agranulocytosis due to (ab), 896
Prostate gland, bone marrow studies in carcinoma of (ab), 709
lymphosarcoma of (ab), 172
myelofibrosis and generalized osteosclerosis secondary to carcinoma of (ab), 172
Prostatic urethra, cystourethrography in diagnosis of diseases of, 640
Protection for high energy radiation and radioactive isotopes, physical methods of achieving (ab), 722
from ionizing radiations from high voltage roentgen rays and radioisotopes, physical, cytological and medical aspects of (ab), 721
Pruritus due to Hodgkin's disease, effect of adenylic acid therapy on (ab), 896
Pseudohypoparathyroidism (ab), 875
Psoriasis, arthritis in, 635
Pterion, hyperostosing meningioma of (ab), 1025
Pyelography, retrograde, evaluation of in management of renal trauma (ab), 699
retrograde, results with use of new contrast medium in (ab), 340
retrograde, usefulness of, in diagnosis of calculi retained in ureter (ab), 171
Pyelonephritis, acute necrosis of renal papillae in (ab), 702
Pyelorenal cysts (ab), 700
Pylorus, duodenal ulcers simulating prepyloric lesions, 603
hypertrophy of, in adult (ab), 1044
hypertrophy of, in infants and in adults, genetic connection between (ab), 691
prolapse of gastric mucosa through (ab), 332
Pyopneumohepatitis, 785

Q fever, roentgen manifestations of (ab), 323

Radiation, acute injuries from, caused by atomic bomb (ab), 879
atomic bomb, radioresistant cells in certain radiosensitive tissues of swine exposed to (ab), 370

atomic, effects of, on oral and pharyngeal mucosa (ab), 721
dosage, clinical, interrelation of time, area and therapeutic ratio (ab), 371
effects, cell viability, and induced resistance to malignant growth, relation between (ab), 557
exposures from use of shoe-fitting roentgenoscopes (ab), 374
high energy, physical methods of achieving protection for (ab), 722
injury in radiologic work (ab), 889
injury to amphibian cell nuclei, nature of (ab), 373
scattered, from fluoroscopic units in 15 institutions, survey of (ab), 188
sickness, new drug in treatment of (ab), 181
syndrome, acute, in man (ab), 879
therapy, present status of in leukemias (ab), 184
Radiations, ionizing, from high voltage roentgen rays and radioisotopes, physical, cytological and medical aspects of protection from (ab), 721
mixed, tentative dose units for (ab), 906
Radioactive impurities, detection of, by constant solubility test (ab), 364
isotopes, physical methods of achieving protection for (ab), 722
materials, localization of, in phantom brain (ab), 554
silver, in vivo localization of, at predetermined sites in tissues, 831
sources, linear, improved dose calculator for (ab), 898
substances, experimental determination by ionization methods of rate of emission of beta- and gamma-ray energy by (ab), 186
tracers, detection of cretinism by (ab), 894
tracers, diagnostic uses of, in infancy and childhood (ab), 894
Radioautography, concentration of I^{131} in thyroid cancer, shown by (ab), 363
detail and survey (ab), 187
Radiodermatitis and necrosis (ab), 183
Radiogold in treatment of pelvic cancer, 75
Radioiodine in diagnosis of lingual thyroid (ab), 186
in thyroid gland, external measurement of (ab), 186
uptake curves in humans (ab), 362
Radioisotopes, course in instrumentation for, 307
in research, courses in techniques of using, 663
new laboratory for research on, 308
physical, cytological and medical aspects of protection from ionizing radiations from (ab), 721
Radiological Conference, Mid-Winter, 1020
health, inservice training course in, 1020
Society of New Jersey, 664
Radiology in medical school, 795
some aspects of, in war-time Germany (ab), 187
Radiophosphorus See also Phosphorus, radioactive
determination of blood volume in heart and lungs and cardiac output through injection of (ab), 363
Radioresistance, increased, of red bone marrow after anoxia (ab), 373
Radium applicator, endobronchial (ab), 372
applicator, nasopharyngeal, in treatment and prevention of deafness (ab), 890
containers, dosimetry of (ab), 902
emanation plant, remote controlled (ab), 555
needle therapy in cancer of cervix uteri, 1010
sources, linear, in air and water, isodose measurements of, by means of automatic isodose recorder, 989
Rapidograph, rapid automatic serialization of roentgen-ray exposures by (ab), 372
Rectum, cauda equina syndrome due to carcinoma of (ab), 164
obstructive lesions of (ab), 165
polypoid lesions of (ab), 165
Renal insufficiency, long standing, with extensive metastatic calcifications, 423
Research fellowships of the American College of Physicians, 306
Respiratory gas exchange in anthracite coal miners with pulmonary complaints (ab), 1031
Retention, chronic (ab), 705
Reticuloses, pulmonary changes in (ab), 1038
Retinoblastoma, roentgenographic aid in diagnosis of, 735
Rheumatic fever, management of, and early complications (ab), 1040
Ribs, giant cell tumor of (ab), 868
Rickets, refractory, 590
vitamin D resistant, 590
Roentgen apparatus for intense roentgen radiation of short duration (ab), 889
cinematography, apparatus and technique for demonstration of cardiac chambers and great vessels by (ab), 904
cinematography in congenital heart disease (ab), 530
diagnosis, generalized subserous emphysema through a single puncture, 723
diagnosis of free intra-abdominal gas, prone position in, 778
examination, conventional, in operable congenital heart disease (ab), 328
irradiation, combination of clinical therapy with (ab), 353
irradiation of small area, thymic death following (ab), 355
manifestations of Q fever (ab), 323
periscope, new type of, for intracavitary therapy, 492
-ray exposure, rapid automatic serialization of, by rapidograph (ab), 372
-ray film badge dosimeter, calibrated, 475

-ray surveys of chest in general hospitals (ab), 317
rays, biologic dosage of (ab), 881
rays, fetal tolerance to (ab), 888
rays, increase of biologic effect of, by ultrasonic waves (ab), 556
rays, individual differences in sensitivity of human skin to (ab), 355
rays, method of concentrating, for deep therapy (ab), 889
rays, physical, cytological and medical aspects of protection from ionizing radiations from high voltage (ab), 721
rays, potentialities of synchroton accelerator as generator of, for medical use (ab), 371
rays, protection of living organism against, by chemical means (ab), 904
rays, toxicity and pathological effects of, in chicken, 289
rays, toxicity of, to chick embryo, 280
rays, 20 million volt, biological evaluation of, 963
survey procedures, reliability of, 32
therapy, supervoltage, clinical usefulness and limitations of (ab), 354
unit for planar roentgenography in vertical, oblique and horizontal positions (ab), 903
ROENTGENOGRAM, preliminary, of abdomen in diagnosis of congenital obstruction of intestine, 430
ROENTGENOGRAMS of colon after colostomy, method for improving, 835
use of, in assessing disproportion of pelvis (ab), 553
ROENTGENOGRAPHY, conventional, in diagnosis of cardiovascular anomalies (ab), 328
planar, examination in pleural effusions by (ab), 318
planar, in osteomyelitis (ab), 898
planar, roentgenoscopic preselection of plane of, in chest lesions (ab), 317
ROENTGENOLOGY, neurologic, continuation course in, 306
ROENTGENOSCOPES, shoe-fitting, potential dangers in uncontrolled use of (ab), 374
shoe-fitting, radiation exposures from use of (ab), 374
ROENTGENOSCOPIC preselection of tomographic plane in chest lesions (ab), 317
units in fifteen institutions, survey of scattered radiation from (ab), 188
ROENTGENOSCOPY, place of, in diagnosis of peptic ulcer (ab), 156
table unit for examination of infants by (ab), 1028
ROSSELET, ALFRED, 1887–1950, obituary, 842
portrait, 842

SANTE, L. R., honored, 1020
SARCOIDOSIS, clinical and roentgenological study (ab), 1032
SARCOMA of cervix (ab), 361
180, effects of nitrogen mustards on (ab), 366
osteogenic, aneurysmal bone cyst mistaken for (ab), 867
osteogenic, as complication of osteitis deformans (ab), 175
polypous, of esophagus (ab), 537
synovial, of chest wall (ab), 1028
SCALP, hairy, tumors of (ab), 672
Sciara, resistance of, to mutagenic effects of irradiation (ab), 371
SCIATICA, air and pantopaque myelograms in diagnosis of, from disc herniation (ab), 342
SCLERODERMA, pulmonary manifestations of (ab), 679
SCLEROSING conditions of bone (ab), 869
SEABRIGHT bantam syndrome (ab), 875
SECRETORY studies on isolated stomach (ab), 900
17-KETOSTEROIDS, neutral, in rheumatoid arthritis and spondylitis (ab), 709
SHOE-FITTING ROENTGENOSCOPES, potential dangers in uncontrolled use of (ab), 374
radiation exposures from use of (ab), 374
SHOULDER pain, with particular reference to "frozen" shoulder (ab), 176
painful, significance of roentgen changes in upper end of humerus (ab), 177
SICKLE CELL disease simulating osteomyelitis (ab), 872
SIGMOID diverticulitis, symptoms of obstruction from (ab), 164
volvulus of (ab), 164
volvulus, roentgen picture of (ab), 337
SILICO-TUBERCULOSIS, mortality and survival rates in males with (ab), 320
SILICOSIS, massive hyperplastic granulomatous (ab), 320
mortality and survival rates in males with (ab), 320
SILVER, radioactive, in vivo localization of, at predetermined sites in tissues, 831
SINUS, coronary, drainage of pulmonary veins into (ab), 326
SINUSES, paranasal, osteomas of, and treatment (ab), 671
SINUSITIS, chronic maxillary, pathology, diagnosis and treatment of (ab), 316
SKELETAL age limits of apparently normal six-year-old children, summary of forty-nine radiologists' opinion on, 442
SKELETON, effect of urethane therapy on disease of, 799
Looser-Milkman's syndrome with neurofibromatosis Recklinghausen and general decalcification of (ab), 714
SKIN, beryllium granulomas of, from broken fluorescent tube (ab), 876
human, beta ray burns of (ab), 364
human, individual differences in sensitivity of, to roentgen rays (ab), 355

Skull fracture, mechanism of (ab), 1023
Smokes, toxic, pulmonary effects of (ab), 904
Sodium iodide, radioactive, distribution of radioactive iodine in mice with and without tumor 15091a after injection of (ab), 905
tetraiodophthalicfluorescein for intravenous cholecystography (ab), 166
South Carolina X-Ray Society, 143
Sphenoidal ridge, roentgen diagnosis of meningiomas of (ab), 314
Spinal cord, arterial anomalies of (ab), 341
tumors of (ab), 342
Spine, dorsolumbar, fractures of (ab), 177
normal, mechanism of rotation in combination with lateral deviation in (ab), 716
solitary plasmocytoma of (ab), 177
thoracic, lateral roentgenographic examination of (ab), 716
thoracic, unilateral tuberculous abscess of (ab), 716
Splanchnicectomy, peptic ulcer following (ab), 542
Spleen, calcified cyst of (ab), 553
epidermoid cyst of (ab), 1048
Spondylarthritis in children, 20
Spondylitis, neutral 17-ketosteroids in (ab), 709
Sponge in abdomen, roentgen diagnosis of, 781
Spot film device, new, fully automatic serial (ab), 903
Sprue, symptomatic (ab), 547
Steno's experiment in man as complication in lumbar aortography (ab), 717
Stenosis, congenital, of lower part of ureter, ureterovesicostomy as treatment of (ab), 704
due to caustic substances, roentgen aspects of periesophageal changes in (ab), 330
hypertrophic pyloric, in infant, roentgen examination in (ab), 546
uncomplicated pulmonic (ab), 1041
Steroid compounds, effects of, on various manifestations of rheumatoid arthritis (ab), 175
Stomach, adenomyosis of (ab), 331
benign tumors of (ab), 161
cancer of, electrogastrography in diagnosis of (ab), 539
cancer of, roentgen diagnosis (ab), 690
carcinoids of (ab), 549
cascade (ab), 157
cortical inhibition of motility of (ab), 899
deforming antritis secondary to ulcer about incisura angularis of lesser curvature of (ab), 543
diagnosis of congenital obstruction of, in newborn (ab), 162
diagnosis of lesions near cardia of (ab), 160
differential diagnosis between benign and malignant ulceration of (ab), 332
diverticula of (ab), 159
downward displacement of cardia of (ab), 1042
examination of, by photofluorographic methods (ab), 897
extension of carcinoma of, into duodenum and esophagus (ab), 544
hourglass, caused by annular muscular hypertrophy (ab), 331
in tuberculosis (ab), 691
isolated, secretory studies on (ab), 900
mucosa of, prolapsed into duodenum, etiology and pathogenesis of (ab), 693
normal and operated, study of, with very warm opaque meal (ab), 543
perforated carcinoma of, simulating perforated gastric ulcer (ab), 544
redundant mucosa of, prolapse of, into duodenum (ab), 692
right-sided, associated with eventration of diaphragm simulating hydropneumothorax, 50
roentgen aspects of diverticula of (ab), 335
roentgen findings in ulcerated neoplasms of (ab), 690
roentgen study of cardia of (ab), 331
sclerosing carcinoma of (ab), 162
studies on physiological chemistry and clinical significance of urease and urea with special reference to (ab), 157
submucosal lipoma of, interesting roentgen sign in, 938
symptomatology and diagnosis of cancer of (ab), 1042
tuberculosis of (ab), 158; (ab), 691
ulcer of, in childhood, 765
Stratigraphy See Roentgenography, planar
Sturge-Weber syndrome (ab), 391
Subluxation of atlanto-axial joint (ab), 173
Sulfate, labeled inorganic, in diagnosis of cartilaginous tumors and their metastases (ab), 186
Sulphur dioxide fumes, pneumoconiosis after exposure to (ab), 320
Synchroton accelerator, potentialities as generator of roentgen rays and electrons of 10–50 mev. energies for medical use (ab), 371
Synovial osteochondromatosis (ab), 178; (ab), 348
Syssomus sternodymus, roentgen diagnosis (ab), 698

Taussig's syndrome (ab), 531
Technique for bronchial instillation (ab), 321
Temporomandibular arthrosis (ab), 342
joint, diseases of, differential diagnosis and treatment (ab), 518
joint, improved roentgenographic technique for (ab), 897
Testicular lesions in mice following parenteral administration of nitrogen mustards (ab), 365
Tetra-ethyl ammonium bromide, effect of, on renal pelvis and ureter (ab), 557
Thorium dioxide as contrast medium in roentgen examination of defunctioned colon after ileostomy (ab), 164
Thrombosis, arteriosclerotic, reconstruction of femoral artery for (ab), 874

intra-auricular, diagnosis of, in living (ab), 530
mesenteric, roentgen examination in, 610
multiple venous, and visceral carcinoma (ab), 550
of internal carotid artery (ab), 313
THROMBOTIC obliteration of aortic bifurcation (E), 496
THYMIC death following roentgen irradiation of small area (ab), 355
THYMUS GLAND, cystic tumor of region of (ab), 1029
THYROID, lingual, radioactive iodine in diagnosis of (ab), 186
THYROID GLAND, cancer, concentration of I^{131} in, shown by radioautography (ab), 363
carcinoma of (ab), 358
carcinoma of, of base of tongue (ab), 519
comparative studies of short and long distance counting of radioactivity of (ab), 362
external measurement of radioiodine in (ab), 186
evaluation of radioiodine measurements made with counter tube in apposition to isthmus of (ab), 362
tumors, preoperative estimation of function in (ab), 362
what nodules of, are to be feared (ab), 362
TIBIAL plateau, fractures of (ab), 864
TISSUES, in vivo localization of radioactive silver at predetermined sites in, 831
soft, simultaneous calcifications of, associated with multiple destructive processes of phalanges of hand, 649
TOMOGRAPHY See Roentgenography, planar
TONGUE, carcinoma of thyroid of base of (ab), 519
TOXICITY of roentgen rays in chicken, 289
of roentgen rays to chick embryo, 280
TOXOPLASMIC defects, roentgen diagnosis of (ab), 1024
TOXOPLASMOSIS, roentgen semeiology of (ab), 719
TRACHEA, fatal interstitial and mediastinal emphysema following accidental needle perforations of, during jugular venipuncture (ab), 527
tuberculosis of (ab), 321
TRAPEZIO-METACARPAL joint, osteoarthritis of (ab), 344
TRAPEZIUM, congenital abnormality of (ab), 344
TRAUMA, head, roentgenologic aspects of, 399
TUBERCULOSIS, aseptic necrosis of femoral head simulating (ab), 862
diagnosis of pneumonia preceding (ab), 525
endobronchial, and physiopathology of cavities (ab), 1035
gastric (ab), 158
generalized, generalized hemangio-sarcomatosis erroneously considered as (ab), 1034
implications of changing morbidity and mortality rates from (ab), 679
incidence of tuberculous bronchitis in pulmonary resections for (ab), 1034
of bronchial tree (ab), 1036
of knee joint in children, differential diagnosis of (ab), 715
of stomach (ab), 691
of trachea and major bronchi (ab), 321
of uterine cervix, radiation treatment of (ab), 887
pulmonary, calcification in, 747
pulmonary, comparative study of results of examinations of population at large with results of examinations of tuberculosis contact cases in detection of (ab , 520
pulmonary, in children, excisional surgical treatment of (ab), 680
pulmonary, prevalence of clinically significant, among inmates of New York State penal institutions (ab), 679
pulmonary, Rasmussen's aneurysms and fatal hemorrhage in (ab), 320
stomach in (ab), 691
TUMOR, brain, cerebral angiography in (ab), 1027
cystic, of thymic region and myasthenia gravis (ab), 1029
giant cell, aneurysmal bone cyst commonly mistaken for (ab), 867
giant cell, of ribs (ab), 868
of carotid body associated with nodular goiter (ab), 676
TUMORS, alveolar cell, of lung, 1
benign bronchial, clinical observation of (ab), 152
benign, of esophagus (ab), 688
benign, of stomach (ab), 161
brain, clinical use of radioactive phosphorus in surgery of (ab), 364
brain, diagnosis of, and other space-occupying diseases (ab), 314
carcinoid (ab), 334
cartilaginous, and their metastases, labeled inorganic sulfate in diagnosis of (ab), 186
chemotherapy of (ab), 365
malignant, chemotherapy of (ab), 369
malignant, of parathyroid gland (ab), 1029
malignant, production of, in rats, with radioactive phosphorus (ab), 905
malignant, study of increased differentiation in, from irradiation (ab), 881
mediastinal, of blood vascular origin (ab), 329
metastasizing cerebellar (ab), 517
metastatic pineal (ab), 1025
nasopharyngeal (ab), 315
of bladder (ab), 172
of bone, diagnosis of (ab), 349
of brain complicating pregnancy (ab), 674
of carotid body (ab), 1028
of forehead and hairy scalp (ab), 672
palpable, of gastrointestinal tract in region of small pelvis, roentgen visualization of (ab), 544
rate, of esophagus (ab), 537
spinal cord (ab), 342

thoracic, angiocardiography for investigation of (ab), 323
thyroid, preoperative estimation of function in (ab), 362

Ulcer See also Peptic ulcer
gastric and duodenal, relationship of frequency of (ab), 1043
gastric, in childhood, 765
Ulceration, benign and malignant, of stomach, differential diagnosis between (ab), 332
Ulcers, duodenal, simulating prepyloric lesions, 603
Mann-Williamson, failure of vagotomy to prevent formation of, in dogs (ab), 904
radionecrotic, fluorescein as adjunct in treatment of (ab), 183
Ultrasonic vibration, effect of, on formed elements of blood from normal and leukemic subjects (ab), 902
waves, increase of biologic effect of roentgen rays by (ab), 556
Urease and urea, physiological chemistry and clinical significance of, with special reference to stomach (ab), 157
Ureter, primary carcinoma of (ab), 340; (ab), 703
retrograde pyelography in diagnosis of calculi retained in (ab), 171
ureterovesicostomy as treatment of congenital stenosis of lower part of (ab), 704
Ureteral orifice, ectopic (ab), 704
Ureterostomy, use of polythene and polyvinyl tubing in (ab), 341
Ureterovesicostomy as treatment of congenital stenosis of lower part of ureter (ab), 704
Urethane in treatment of mycosis fungoides (ab), 897
therapy, effect of, on multiple myeloma, 799
treatment of multiple myeloma with (ab), 896
Urethra, calculus of, in male (ab), 703
prostatic, cystourethrography in diagnosis of diseases of, 640
stricture of, in children (ab), 173
Urethrography, double contrast (ab), 173
fatal embolism following (ab), 701
Urology award, 505
Uterus, adenocarcinoma of body of (ab), 891
new diagnostic adjunct for cancer of (ab), 169

Vagina, radium therapy of primary carcinoma and other malignant lesions of, 86
Vagotomy, complete, recurrence of gastric ulcer after (ab), 542
failure of, to prevent formation of Mann-Williamson ulcers in dogs (ab), 904
limitations of, in treatment of peptic ulcer (ab), 542
Valsalva maneuver, use of, in contrast visualization of aorta and great vessels (ab), 530
Varices, post-thrombotic, results of phlebography and radical operative treatment (ab), 179
Vascularization of aorta (ab), 534
Vein, great cerebral, arteriovenous fistula of (ab), 517
Veins, pulmonary, drainage of, into coronary sinus (ab), 326
Vena cava, superior, congenital aneurysm of (ab), 1040
Venography, proximal femoral (ab), 873
role of, in mediastinal and paramediastinal lesions (ab), 534
Vertebra plana osteonecrotica, study of (ab), 351
Vertebrae, lumbosacral, anomalies of, in 550 individuals with symptoms referable to low back, 624
Visceral survey, fast roentgen (ab), 689
Vitamin B_{12}, bacterial synthesis of, in alimentary tract (ab), 879
D resistant rickets, 590
Volvulus of sigmoid (ab), 164
sigmoid, roentgen picture of (ab), 337
Vomiting in infants, cardio-esophageal relaxation as cause of (ab), 1030
von Recklinghausen's disease, sarcomatous transformation of (ab), 879
Vulva, carcinoma of (ab), 359; (ab), 360

Washington State Radiological Society, 143
Whipple's intestinal lipodystrophy (ab), 547
Wilms' tumor, review of five year survivals (ab), 358

Yaws (ab), 350

Zirconium, compounds of, for x-ray media (ab), 370

AUTHOR INDEX TO VOLUME 63

* = Original Article

Abbott, O. A., 1040
Abbott, O. D., 713
Abeshouse, B. E., 704
Abrahamsen, Harold, 704
Abrams, Bernard, 1048
Ackerman, Alfred J., 1032
Ackerman, Lauren V., 847
Adams, W. E., 319, 688
Aguirre, Fidel, 665
Ahmann, C. F., 713
Åkerlund, Åke, 696
Albee, R. D., 159
Albertengo, Juan B., 707
Albot, Guy, 543
Alexander, Carter M., 361
Alexander, G. H., *940
Alfaro, Abel, 708
Alfaro, Samuel, 708
Allen, L. M., 166
Allen, Willard M., *75
Almklov, J. R., 872
Alpert, L. K., 894
Alyea, E. P., 709
Andersen, K., 345
Andersen, S. Ry., 881
Anderson, A. F., 698
Andler, Maxwell, 674
Andrews, Gould A., *61
Andrews, J. Robert, 850
Andrus, William DeW., 686
Antoni, Nils, 717
Anzilotti, Alberto, 884
Apley, J., 531, 687
Ardran, G. M., 904
Arendt, Julian, 530, *950
Arias Bellini, M., 164
Ariel, I. M., 1037
Arkin, Alvin M., 716
Arnell, Sigfrid, 855
Arneson, A. N., 93, 112
Arons, Walter, 155
Ash, J. E., 172
Ashkenazy, Moses, 182
Ask-Upmark, Erik, 689
Aub, J. C., 897
Auerbach, O., 321
Avery, E. E., 1037

Babaiantz, L., 719
Babkin, B. P., 899
Bachi, Sergio, 519
Bachman, Arnold L., 356
Baclesse, F., 357
Baeyer, Eric V., 162
Baggenstoss, Archie H., 550
Bagnall, D. J. T., 320
Bailey, Wilbur, *501
Baker, A. H., *810
Baldwin, Maitland, 706
Ballantyne, J. Chalmers, 316
Balli, Luciano, 349
Bamatter, F., 719
Banfield, W. G., 365
Barden, Robert P., 517
Bargen, J. Arnold, 886
Barnes, Alan C., 850
Barnett, J. C., 556
Barry, Frank McA., 545
Bass, H. E., 720
Batalla Sabaté, Luis, 171
Bates, G. C., 701
Bauer, Richard D., 366
Baum, George L., 553
Baxter, O. D., 841
Beau, A.-M., 697
Becker, Norman O., 1039
Bedoya, J., 887
Beecham, Clayton T., 93, 112
Beeler, John W., 181
Begley, Joseph W., Jr., 671
Beierwaltes, W. H., 362
Beilly, Jacob S., *53
Bell, John L., 539
Bell, Joseph C., 848
Beller, Aaron J., 341
Benassi, Enrico, 333
Bengloff, Harold, 339
Berenberg, W., 1030
Berens, David L., *391
Berger, H., 903
Berger, S. M., 849
Berke, R., 720
Berman, Doreen, *974
Bernier, Joseph L., 721
Berning, Hermann, 152
Bernstein, C., 695
Bersack, Solomon R., *624
Berven, Elis G. E., 359
Bethell, Frank H., *61
Bétoulières, P., 184
Bettman, Ernest H., 347
Bickel, Georges, 369
Bierman, Howard R., 367, 902
Bigelow, Robert, 696
Bigger, I. A., 186
Bingold, A. C., 713
Binkley, F.M., 523
Bishop, R. E., 716
Bissolino, Gerolamo, 529
Bissonnette, R. P., 159
Bjerkelund, C. J., 547
Black, J. I. Munro, 315
Black-Schaffer, B., 547
Blades, Brian B., 526
Blake, Paul S., *816
Blaney, Loren F., 364
Bloch, Henry, 370
Blumel, Johanna, 362
Board, Francis A., 368
Body, A. M., 180
Bogoch, A., 527, 1041
Bohatirchuk, F., *649
Boldrey, Edwin, 517
Bondani, Luigi, 340
Bonoff, Chester P., 1034
Bonte, Frederick J., 905
Boone, Bert R., 326
Borst, W. H., 176
Bosch del Marco, Luis M., 157
Botti, G. D., 329
Boucot, Katherine R., 317
Boutelle, W. E., 871
Bowers, John Z., 879
Bowing, Harry H., *86
Boyd, George A., 368
Bragg, E. A., Jr., 1033
Braggion, P., 672
Brahms, S. A., 686, *819
Braibanti, T., 340, 898
Brailsford, James F., 349, 869
Brauer, R. O., 183
Breitländer, K., 355
Brewer, Lyman A., 1037
Bricker, E. M., 873
Bridgman, B. A., 847
Broderick, T. C., 1047
Broderson, H., 372
Brombart, M., 1030
Bromer, Ralph S., 29
Brown, C. H., 159
Brown, James Barrett, 850
Brown, P. W., 362
Brussell, Irvin J., 518
Buchman, Joseph, 872
Buchtala, V., 866
Buckles, Maurice G., 868, 1034
Bürger, Hermann, 157
Buie, L. A., 181
Bulbulian, Arthur H., 855
Bull, James W. D., 513
Bullo, Enzo, 509
Bunts, Alexander T., 675
Burchell, Howard B., 855
Burk, Lloyd B., Jr., 175
Burkell, C. C., 357
Burman, Michael, 871
Burt, James C., 169
Buschke, Franz, 354

Buser, Julian W., 1046
Buzzi, Germano, 164

Caffaratti, Enrico, 541
Caffey, John, 547
Calder, E., 317
Callendine, George W., 850
Cames, Oscar J., 521
Camp, E. H., 542
Camp, Raymond, 546
Campbell, E. H., 1025
Campbell, J. A., 848
Campbell, M. F., 173
Campbell, R. J. C., 331
Cantor, Meyer O., 507, 694, *959
Cantrell, James R., 370
Cantril, Simon T., 354
Cardon, Leonard, 530
Carlquist, John H., 163
Carmody, Morris G., 876
Carothers, E. L., 720
Carp, I. J., 703
Carpender, J. W. J., 851
Carrié, C., 902
Carroll, David S., 716
Carter, Ray A., 323
Casalonga, 321
Case, James T., 777
Cassel, M. A., 681
Cassels, D. E., 319
Castellanos, Agustin, *255, 534
Castigliano, S. Gordon, *492, *968
Cattell, R. B., 694
Caulk, Ralph M., 464
Cave, Edwin F., 345
Celander, Hjöordis, 363
Cesanelli, Alfredo, 521
Cesarani, Fausto, 690
Cevese, P. G., 672
Chaffee, J. S., 515
Chamberlain, Richard H., 554
Chandler, Fremont A., 866
Chassin, J. L., 163
Chi, Paul S. H., 688
Chiray, M., 506, 696
Chitwood, James L., 674
Christopherson, E. F., 1041
Christopherson, William M., 868
Chusid, J. G., 515
Clagett, O. Theron, *1, 329, 1035
Clark, Dwight E., 696
Clay, Richard C., 690
Clifford, J., *475
Clifford, William J., *414
Cliffton, Eugene E., 536
Cocchi, Umberto, 696, 714
Coe, F. O., 254, 848
Cohen, H. L., 556
Cohen, Ira, 341
Cohen, Lionel, 371
Cole, W. H., 358
Collins, E. G., 181
Collins, Martha D., 1034
Collins, W. T., 544
Colsen, Kurt, 174
Congiu, Aldo, 551
Coodley, Eugene L., 688
Cooper, David A., 317
Cooper, George, Jr., 156
Cope, O., 362
Coppedge, Thomas O., 850
Coray, Q. B., 163
Corbett, Austin J., 676
Cornwall, C. R., 166
Corrigan, K. E., 850, 894
Cosby, Lewis, Jr., 156
Cottet, Pierre, 537
Cowing, Russell F., 188
Cozen, Lewis, *277
Cozza, Francesco, 557
Craig, A., 1027
Craig, M. S., Jr., 181
Craig, W. McK., 341
Crawford, H. B., 847
Crawford, Vernon, 186
Cronin, T. D., 183
Crothers, B., 1025
Crouse, Helen V., 371
Culbreth, George G., 1027
Culver, Gordon J., *938
Cunningham, B. T., 681
Cunningham, J. N., 877
Curr, J. F., 348
Curry, Robert W., 1027

Dabbs, G. H., 174
Dahlin, David C., 718
Dale, W. Andrew, 1046
Dalla Bernardina, Lino, 543
D'Alò, Roberto, 509
D'Angio, Carl J., 711
Daniel, William H., 165
Danis, André, 549
Dannenberg, Max, *53
Danopoulos, Ev., 180
Dargeon, Harold W., 877
Daubresse, E., 184
Davidoff, Leo, 514
Davis, Loyal, 182
Davis, R. H., *475
Davis, Thomas J., *409
Davison, Forrest Ramon, 510
Deamarais, M. H. L., 709
Debray, C., 696
de Carvalho, Lopo, 684
Decker, David G., *86
de Gutiérrez-Mahoney, C. G., 515
Delattre, P., 696
DelDuca, V., 877
De Martini, Luiz Carlos, 157
de Mello, R. F., 522
de Moraes, Clóvis Cardoso, 528
De Nardi, Joseph M., 876
Denlinger, Ross B., 323
Denneen, E. V., 1047
Derennes, R., 344
Deriu, Giuseppe, 538
Desneux, J.-J., 551
Deucher, W. G., 548
Dexter, L., 1041
Diamond, Israel, 336
Dickey, Lloyd B., 1031
Dickson, E. D. D., 184
Didier, Arnoldo E., 710
Dietz, M. Wendell, 322
Dixon, Claude F., 886
Dobrak, Alfred, *938
Dobrina, D., 342
Dobyns, B. M., 362
Dockerty, Malcom B., 338
Dodge, Mark, 341
Doll, Richard, 544
Dolley, Frank S., 1037
Domínguez Quesada, Serafín 540
Domzalski, Casimir A., Jr., 166
Donovan, G. E., 187
Donzelot, E., 155
Dos Santos, Reynaldo, 712
Dotter, Charles T., *222, 686, 849
Douzelot, E., 687
Dow, J. W., 1041
Dragstedt, L. R., 542, 900, 904
Dreisinger, F., 1027
Drolet, Godias J., 679
Dry, Thomas J., 855
Düben, W., 867
Duffy, Mary, 175
Dunbar, Howard S., 849
Dunner, Lasar, 320
Dunphy, J. E., 876
Duryee, William B., 373
Dyke, W. J. C., 879

Eaton, Robert M., 899
Ecker, Arthur, 1025
Edgren, Walter, 714
Edmeades, Donald T., 1038
Edwards, Harold C., 335
Edwards, Herbert R., 679
Edwards, Jesse E., 855
Effler, Donald B., 18
Eggimann, Pierre, 524
Eibach, Ernst, 1042
Eickhoff, Wilhelm, 506
Eisenberg, F. F., 366
Eisenberg, R. B., 1028
Eklund, Lennart, 889
Elkeles, A., 332, 532
Elkin, M., 1041
Ellinger, A. J., 875

Ellinger, F., 556
Ellinger, George F., 326
Elliott, Raven Rivera, *831
Ellis, Frank, 355, 360
Ellman, Philip, 709
Elmore, Francis H., 901
Elsey, Edward C., 847
Elvidge, Arthur R., 313, 706
Emam-Zade, A-M., 155
Eman-zade, Am., 687
Enersen, D. M., 904
Engelstad, Rolf Bull, 897
Entin, Juan, 1045
Eppinger, E. C., 1041
Epstein, B. S., *265, 556, 1041
Epstein, Joseph, *974
Epstein, Joseph A., 341
Erber, Bruno, 1035
Erf, Lowell A., 366
Ernberg, Torsten, 549
Ernst, Edwin C., 849
Escalle, J. E., 155
Esser, Claus, 676
Estes, Ambrose C., 519
Ettinger, Alice, *603
Evans, Arthur T., 847
Evans, Byron H., 1037
Evans, E. Mervyn, 343
Evans, F. Gaynor, 708
Evans, John A., *375
Evans, John W., 716
Evans, W. A., 848
Eversole, Joseph W., 877

Failla, G., *489
Fairbank, H. A. Thomas, 346, 870, 878
Falk, Mortimer S., 524
Farr, Hollon W., *1010
Fawcitt, Richard, 335
Feiring, Emanuel, 514
Feldman, Maurice, 540
Felson, Benjamin, *196, 334, *740, 847
Ferdman, José, 1045
Ferris, B. G., 1041
Figley, M. M., 328
Fischl, Jacques R., *42
Fisher, Hyman, 1034
Fitts, William T., Jr., 347
FitzGerald, Oliver, 157
Fitzgerald, P. J., 334, 363
Fitzgibbon, J. H., 160
Fleischner, F. G., 528, 695
Fletcher, Gilbert H., *95, 1021
Flink, E. B., 172
Foged, Jens, 342
Foote, Frank W., Jr., 363
Forrai, Elemér, 863
Forrester-Wood, W. R., 693
Forsee, J. H., 1033
Forssberg, Arne, 904
Fossati, Franco, 665
Foster, John, *391
Foussier, B., 368
Fracasso, E., 330
Frankel, Kalman, 1027
Franklin, R. H., 1029
Franklin, Wm., 331
Frantzell, Arne, 689
Freeman, Bromley S., 183
Freeman, N. E., 530
French, Lyle A., *816
French, R. B., 713
Fricke, Robert E., *86
Friedell, H. L., *559, *576, 905
Friedman, Arnold P., 514
Friedman, Milton, 359, *989
Friend, F., *810
Frimann-Dahl, J., *610
Fritz, J. M., 319, 542
Frobese, A. S., 333
Fromm, Günther A., 888
Fry, D. W., 371
Fryer, Minot P., 850
Fuchs, K. H., 866
Fulghum, Charles B., 1029
Fullenlove, T. M., 530, 707
Fulton, Harold, *735
Furcolow, Michael L., 321
Fust, J. A., 340
Futcher, Palmer H., 896

Gaensler, E. A., 895
Gahagan, H. Q., 170, 358
Galizzi, João, 551
Gallone, Patrizio, 665
Gálvez Armengaud, F., 708
Garcia, Manoel F., 157, 338
Garcia, Otto, *255
Gardner, W. James, 253
Garland, L. H., *32
Gass, H., 1027
Gaudin, H., 701
Gavey, C. J., 327
Geist, Robert M., Jr., 675
Gelfand, Micheal, 525
Genovese, P. D., *423
Gentile, A., 703
Gentile, Walter, 550
Gershon-Cohen, J., 849
Ghersi, Julio A., 888
Gianturco, C., 689
Gilfillan, R. S., 530
Gill, Dan C., 364
Gillespie, H. W., 350
Gillick, Frederick G., 326
Gimena, J., 708
Glocker, Richard, 147
Glover, Donald M., 545
Goland, Philip P., 554
Golden, Ross, 144, 655, *769
Goldfeder, Anna, 557
Goldin, A., 365
Goldstein, A. E., 704
Goldstein, Philip, 527
Goldstein, Stanton L., 182
Goluboff, Nathan, 869
Gomberg, H. J., 362
Good, C. Allen, *1, 322
Goodale, Raymond H., *414
Goodale, W. T., 1041
Gordon, A. J., 686, *819
Gordon, B., 1031, 1032
Gordon, Everett J., 179
Gordy, Phillip D., 518
Gore, Ira, 155
Gotten, Nicholas, 513
Goutkine, J., 1030
Govea, Juan, 665
Granjon, A., 697
Grau, E., 883
Gray, L. H., 185
Grebe, Leonhard, 148
Green, H., 687
Green, John R., *391
Greenman, L., 874
Greenspan, E. M., 894
Greiberg, Joseph A., 179
Grier, G. W., 499
Griffin, Mark A., Jr., 554
Griffith, Eugene R., *1
Griffith, George C., 1038
Griffiths, W., 702
Gross, Robert E., 533
Gross, Robert J., 164, *399
Grout, J. L. A., 335
Grünberger, V., 1048
Grunow, Otto H., 847
Guardabassi, Luigi, 718
Guerreiro, Geraldo, 716
Gunderson, R. W., 856
Gurdjian, E. S., 1023
Gurian, G., 330
Gustad, Viktor, 702
Gutmann, Helmut R., 364
Gutmann, R. A., 506

Hadley, Lee A., 848
Hagelstam, Lars, 144
Hahn, P. F., 720
Haimovici, Henry, 873
Hallberg, Olav E., 671
Hamlin, E., Jr., 362
Hamil, Brenton M., 1035
Handler, P., 547
Hanelin, Joseph, 847
Hannan, J. R., *239, 342
Hansen, A. E., 872
Hardy, R., 320

Harnasch, Hans, 553
Harper, Paul V., Jr., 900
Harrington, D. R., 849
Harrington, Stuart W., 329
Harrington, William J., 896
Harris, Milo, 156
Harris, R., 534
Harris, W. Robert, 862
Harris, William, 356, 465, 928, 1009
Harrison, B. J., 30
Harrison, F. G., 340
Harrison, Stewart H., 177
Hartley, J. Blair, 168
Hartweg, Helmut, 680
Hartz, Alvin S., 525
Harvey, W. P., 1041
Haubrich, R., 155, 358
Hawkes, C. D., 513
Haworth, James B., 332
Hawthorne, H. R., 333
Hayden, H. S., 850, 894
Hayes, Edward W., 507
Haymaker, W., 1025
Haynes, F. W., 1041
Heacock, C. H., 525
Head, J. R., 1037
Healy, M. J., Jr., 542
Heaton, T. G., 1037
Heberer, G., 1039
Heffernon, Elmer W., 159
Hefke, Hans W., 546
Heim de Balsac, R., 155, 687
Heinen, John H., Jr., 174
Hellbaum, Arthur, 175
Hellems, H. K., 1041
Hellerstein, H. K., 530
Helmke, Roderich, 355
Helmsworth, James A., *196
Hempelmann, L. H., 374
Hempelmann, Louis, 364
Hemphill, Arthur C., 537
Hendrick, J. W., 887
Hendrix, J. P., 547
Henkin, William, 165
Henschen, Folke, 509
Herdman, J. P., 906
Hermann, Jack, 879
Hermel, M. B., 849
Hernández Gonzalo, P., 522
Hernberg, C. A., 155, 714
Hertig, Arthur T., 339
Hertzberg, Johan, 702
Hicken, N. F., 163, 166
Hill, R. F., 363
Hind, H. G., 879
Hine, Gerald J., *989
Hinkel, C. L., *617
Hinton, J. W., 163
Hirsch, Wolfgang, 867
Hirst, John C., 170
Hobbs, Arthur A., Jr., 888
Hochberg, L. A., 1033
Hodges, F. J., 221, 328, 667, 1042
Hodges, P. C., 167, 898
Hodson, C. J., 521
Hoff, H. E., 187
Hogness, John R., 364
Holden, William D., 874
Holinger, P. H., 322
Holland, R. H., 337
Hollender, L., 323
Holman, Cranston W., *222
Holt, John F., *590, 667
Holtham, W. H., 705
Holthusen, H., 668
Hope, John W., 314
Hopkins, William A., 534
Hopkirk, J., 362
Horn, R. C., 1028
Horn, R. H., 874
Horrax, Gilbert, 341, 1026
Hoster, Herman A., *913
House, Ralph W., *442
Howard, H. H., 705
Howard, W. L., 526
Howard, William P., 120
Howarth, J. Cornall, 675
Howes, William E., 849, 911
Horwitz, Thomas, 865
Howorth, M. Beckett, 179
Hudson, G. W., *409
Hughes, C. Robert, *239, 342
Hughes, E. S. R., 863
Huizinga, E., 688
Hultberg, Sven, 889
Hunt, Howard B., *446
Hunter, S. W., 370
Hunter, W. C., 324

Ingelfinger, F. J., 331
Ingram, M. D., Jr., *409, *765
Ioannou, Jean, 317
Isaac, Frank, 152
Ishmael, William K., 175
Israels, Sydney, 700

Jackson, A., 701
Jackson, Chevalier, 677
Jackson, Ira J., 313
Jaco, N. T., 906
Jacobs, Julian E., 178
Jacobson, George, 323
Jacobson, M. B., 703
Jacox, Harold W., 809, 928
Jacquez, John A., *289
James, G. W. H., 180
James, L. A., 1033
James, T. G. I., 313
Janker, R., 530, 904
Jeaffreson, Bryan L., 698
Jenkins, R. T., 898
Jenkins, Robert, 902
Jensik, R. J., 322
Jepson, R. P., 180
Johansmann, Ralph J., 847
Johnson, Alphonso P., *831
Johnson, Charles W., *831
Johnson, Clifford C., 550
Johnson, J. H., Jr., 1044
Johnson, W. F., 329
Johnstone, A. S., 334
Jolly, P. N., 320
Jones, C. M., 164
Jones, Frederick Wood, 866
Jones, George F., *740
Jonsson, S. O., 549
Joplin, Robert J., 847
Josephs, Sydney, 707
Judson, Glenn L., 537
Jutras, Albert, 331

Kaell, Harry I., 866
Kahn, Edgar A., 518
Kalmon, E. H., Jr., 349
Kanter, L., 1037
Kaplan, Abraham, 860
Karnofsky, David A., *280, *289
Katz, Julius, 679
Kay, Saul, 1047
Keibl, Edwin, 893
Kemp, L. A. W., 898
Kennedy, B. J., 897
Kent, Leonard, 894
Kerman, H. D., 851
Kersner, Joseph B., 1046
Key, J. Albert, 860
Kiefer, Everett D., 159
King, J. D., 702
Klein, Armin, 847
Klein, Russell R., 165
Kleinberg, Samuel, 865
Kleitsch, W. P., 329
Kneidel, J. H., *430, 716
Knorpp, C. T., 362
Knowlton, Norman P., 364
Knudtson, K. P., 324
Koebele, F., 323
Köhler, Rolf, 692
Körbler, Juraj, 185
Kofman, O., 877
Koiransky, G. H., *789
Koletsky, Simon, 905
Kondo, B., 1041
Kositchek, Robert J., 694
Kotte, J. Harold, 213
Krämer, W., 161
Krainin, Philip, 711
Kremens, Victor, 849
Kretschmer, Herman L., 171

Kuhn, John F., 175
Kunstadter, Ralph H., 681
Kunz, Heinrich, 903
Kuroiwa, D., 1041

LaDue, John S., 1042
Lahm, W., 881
Laivaag, Kaare, 677
Lampe, Isadore, 362, 522, 667
Land, F. T., 536
Landing, B. H., 365, 366
Lang, L. P., 1031, 1032
Langer, Lazaro, 153
Langston, H. T., 1037
Lanzl, E. F., *963
Larsson, Lars-Eric, 889
Lasserre, C., 344
Lattanzio, Enrico, 318
Lattimer, John K., 849
Lauge-Hansen, Neils, 865
Laurent, Y., 1030
Lawless, Edward C., 868
Lawrence, John H., 361
Lawrence, L. R., 1033
Lawson, Edwin H., 1047
Layton, Laurence L., 186
Ledoux-Lebard, Guy-R., 506
Ledoux-Lebard, R., 506
Lee, F. Wayne, 178
Leifer, Edgar, 364
Leigh, Ted F., 534, *945
Leighton, Robert S., 340
Leiken, S., 851
Lenz, Maurice, 917
León, Juan, 698
Lett, James E., 322
Leucutia, T., 574, 809, 1016
Levene, G., 1033
Levin, R. T., *423
Levine, H. D., 1041
Levinson, David C., 1038
Levitin, Milton, 680
Levrier, Paul, 331
Levy, Silvio, 333, 550
Lewey, Frederic H., 517
Lewis, J. L., Jr., 327
Lewis, K. M., 871
Lewis, Lloyd G., 359
Lewison, E. F., 1028
Leydig, Stanley M., 178
Lichenstein, Louis, 867
Liechti, Adolf, 666
Linde, Paul, 179
Lindell, B., 889
Lindgren, Erik, 717
Lischi, Giancarlo, 320
Lissner, H. R., 1023
Litzow, L. T., 873
Lloyd, Milton Sills, 318
Loehr, William M., *835
Lötsch, Anny, 893
Loevinger, R., *999
Longtin, Marcel, 331
Looney, J. M., 720
Lòpez Engelking, Raúl, 348
Lorman, J. G., 848
Lott, Bruce D., 361
Low, J. R., 156
Lowe, C. E., 875
Lowe, Charles U., 695
Lowell, F. C., 331
Lowell, Lawrence M., 324
Lowery, G. H., 362
Lowsley, O. S., 703
Luce, W. M., *963
Ludwig, J. P., 694
Lura', A., 329
Lustok, Mischa J., 553
Lynch, J. P., 536, 895
Lynxwiler, Chester, 682

Mabbs, D. V., 188
McClintock, John C., 676
McCrea, L. E., 171
McDonald, J. J., 873
McDonald, Ian, 676
McDonald, J. R., 678
McDonald, John R., *1, 153, 322, 329, 341, 1035
McDowell, Frank, 850
McDowell, H. B., 713
MacDuffee, Robert C., 373
McFarland, M. D., 701
McGaw, W. H., 711
McGibbon, J. E. G., 184
MacGillivray, William F., *414
McGuire, Johnson, *196
Mack, J. Keller, 326
McLean, Don W., *959
McMurray, T. P., 864
McNeer, Gordon, 1042
McSwain, B., 349
Maddock, Walter G., 539
Madsen, Valdemar, 682
Magan, M. G., 316
Magnier, Francois, 543
Mahon, H. W., 1033
Maier, Herbert C., 523
Maisin, J., 146
Majarakis, J. D., 358
Maliner, M. M., 1028
Maloof, F., 362
Manalan, M. M., 848
Mandicas, A., 360
Manges, W. B., 1044
Marcozzi, Giovanni, 710
Margules-Lavergne, M. P., 515
Markovits, Emerik, 508
Marks, Hirsch, *470
Marks, J. H., 529
Marley, W. G., 722
Maróttoli, Oscar R., 710
Marquès, P., 184
Marshak, Richard H., 165
Marshall, A. G., 557
Marshall, E. Wayne, 317
Martens, Erich, 715
Martin, John, 182
Martin, W. J., 320
Mason, H. A., 174
Mason, Stephen C., 542
Massie, Edward, 896
Mattoso Filho, S., 173
May, Charles D., 695
Mayer, Charles, 891
Mayer, Léopold, 891
Mayfield, L. H., 336
Meade, Richard H., 526
Megibow, Samuel, *819
Meigs, Joe V., 892
Meinken, Mary Ann, 905
Meissner, W. A., 681
Melamed, Abraham, 693
Mendl, K., 163
Merchán, M., 708
Merrill, E. F., 847
Merritt, H. Houston, 514
Mesa, C. P., 522
Metianu, C., 687
Meyer, John H., 679
Meyer, Ralph R., 372
Meyers, Muriel C., *61
Meyers, William G., 850
Meyler, L., 688
Michelis, Ugo, 1036
Milch, Henry, 863
Miller, A. J., 868
Miller, Earl R., 517
Miller, J. E., 214
Milzer, Albert, 681
Minowitz, W., *999
Miquel Franca, J., 522
Miree, James, 370
Mitchell, G. A. G., 702
Moersch, Herman J., 153
Moir, J. C., 553
Moloney, William C., 896
Montejo, Gustavo, 353
Moore, George E., 166
Moore, Sherwood, 372
Mordeglia, Miguel, 1045
Morgan, Russell H., *189
Morris, John McL., 892
Morrow, Patrice L., 902
Morton, Joseph L., 73, 850, *919
Motley, H. L., 1031, 1032
Moyer, John H., 1032
Mudd, Stuart, 188
Mueller, E. E., 526

Mulvey, Bert E., 342
Munger, A. D., 329
Muri, J., 711
Murison, Paul J., 1042
Murphy, James Peter, *47
Murphy, Philip, 157
Murray, R. C., 863
Musiani, Umberto, 699
Muth, H., 700
Myhre, J., 172

Nachman, Herman M., 186
Naclerio, Emil A., 153
Natelson, S., 556
Nathan, Nestor J. S., 698
Nathanson, I. T., 897
Nathanson, Louis, *467
Nayer, H. R., *50
Neal, W. B., Jr., 900, 904
Neibling, H. A., 338
Neiburgs, H. E., 168
Neligh, Rosalie B., *61
Nemours-Auguste, S., 535
Neptune, Wilford B., 1034
Netherton, E. W., 876
Neuhauser, E. B. D., 30, 695, 1030
Neustadt, Ernest, 871
Newbery, G. R., 187
Newcomer, Victor D., 524
Nichols, Gregory B., *778
Nichols, Russell L., 167, 851
Nicoll, E. A., 177
Nobile, Carlo, 337
Noe, H. A., 365
Nogales, F., 887
Nohrman, Bengt A., 683
Nolan, James F., *75
Norlén, Gösta, 516
North, J. P., 1044
Noyez, D., 712
Nylin, Gustav, 363

Oakes, William R., 364
Obiglio, Julio R., 698
O'Brien, Frederick W., 882
Oddie, T. H., 186
Odell, Lester D., 169
Odell, Richard T., 178
Ogdon, W. E., 1037
Olim, Charles B., 534
Oosthuizen, S. F., 350
Orie, N. G. M., 531
Orkin, L. A., 699
Orlandini, Ivo, 550
Osler, A. Abbott, 534
Oswald, Neville, 1038
Ott, Paul, 886
Ottoman, Richard E., 152
Overholt, R. H., 681
Owen, R. D., 1024

Paaby, Henning, 161
Pack, George T., 1042
Palmer, Eddy D., 691
Palmer, L. H., 165
Palmer, Walter L., 1046
Panzer, Ruth, 539
Pardo Menéndez, V., 522
Park, S. D. S., 536
Parker, Carl P., Jr., 674
Parker, H. M., 906
Parker, Robert L., 855
Parsons, P. B., 848
Paterson, Ralston, 892
Patterson, Priscilla A., *280
Paulson, Donald L., 520
Paunesco-Podeanu, A., 325
Pauzat, D., 344
Pearse, Herman E., 1046
Pearson, C. M., 334
Peck, Dorothea R., 328
Peet, Max M., 518
Pendergrass, Eugene P., 314, *795
Pereira, L. R., 522
Pereiras, Raul, *255, 534
Perkins, Charles W., *473
Pernikoff, M., 1033
Perryman, Charles R., 314
Peters, J. H., 874
Petersen, A. B., 324
Peterson, Gerald M., 848
Peterson, S. S., 894
Phillips, Charles, 519
Pieri, J., 321
Pirkey, E. L., 848, 851
Plenk, Henry P., *635
Plessinger, V. A., 320
Plunkett, Robert E., 679
Pohlandt, Klaus, 1045
Pohle, Ernst A., 146
Pokorná, Lilly, 349
Pollard, H. M., 542
Pollastri, Sante, 699
Popp, Walter C., 181, 886
Poppen, James L., 1026
Portmann, U. V., 589, 851, 890, 918
Potts, A. M., *576
Prather, George C., 666
Prehn, Richmond T., 901
Press, P., 520
Prevedi, Giorgio, 898
Prévôt, R., 1045
Profitt, J. N., 349
Propatorides, J., 158
Prouty, Margaret, 872
Pryde, Arthur W., 550
Psenner, L., 1026
Pund, Edgar R., 168
Quastler, H., 887, *963
Quinlin, Patricia M., 905

Rabwin, Marcus H., 694
Radakovich, Michael, 1047
Ragaglini, Giorgio, 902
Ragan, Charles, 708
Râmniceanu, R., 325
Ramsay, B. H., 681
Rand, Carl W., 674
Randall, David, *640
Ratcliffe, A. Hall, 180
Raulston, B. O., 1041
Raven, Ronald W., 177
Rawls, Noel B., 332
Ray, Bronson S., 849
Redner, B., 545
Reed, E. S., 851
Reed, J. O., 848
Reed, W. K., 170
Reeves, Robert J., *799
Reh, Edward P., 894
Reichel, Wolf S., 356
Reid, F., 167, 339
Reidy, John A., 847
Renfer, H. R., 353
Rennaes, Selmer, 897
Rennie, J. B., 536
Revers, F. E., 176
Reyes, Victor, 1026
Reynolds, L., 850, 894
Rheinlander, Harold F., 336
Rhoades, J. E., 539
Richardson, Fred MacD., 317
Richardson, Harrison H., *940
Richman, Samuel, 850
Richmond, J. B., 875
Ridgway, Lois P., *280
Riding, D., 879
Rienhoff, W. F., Jr., 678
Riese, M. W., 673
Rigler, L. G., 390, 687
Ritter, J. Sidney, 171
Ritter, Vern W., 347
Rivas, M. Ruiz, *723
Rizzo, R. P., 711
Roberts, Brooke, 347
Roberts, Douglas J., *1010
Roberts, M. A., 871
Roberts, M. Hines, 546
Robinson, C. V., 364
Robinson, Franklin, 515
Rodney, Marvin B., *53
Rohr, Karl, 146
Røjel, Kaj, 162
Rogers, H. Milton, 855
Rogers, James V., Jr., *945
Rose, John E., 555
Rosenthal, Sol Roy, 901
Rosselet, Edouard, 351

Rossi, H. H., *489
Roswit, B., 1033
Rothschild, Carl E., 862
Rottino, Antonio, 896
Rowe, A. R., 316
Rudhe, Ulf, 696
Rundles, R. W., 709, *799
Rushforth, A. F., 344
Russell, T. B., 344
Rutledge, David I., 154
Ruttiner, J. R., 180

Sabin, Albert B., 746
Sachs, Allan E., 692
Sachs, M. D., 711
Sadek, H. M., 551
Saenger, Eugene L., *20, 847
Salgueiro, Lidia, 372
Sante, L. R., 237, 849
Santiago-Stevenson, D., 530
Sapin, S. O., 325
Sarre, K., 161
Sarian, J. N., 842
Sauer, P. K., 542
Sawyer, P. N., 539
Scarborough, Robert A., 165
Schack, Jerome A., 373
Scharfe, E. E., 890
Schein, Clarence J., 537
Schenck, Samuel G., 885
Schlesinger, M. J., 529
Schlichter, J., 534
Schmidt, C., 323
Schmidt, Herbert W., 341, 1035
Schnap, Emil H., 849
Schneider, M. D., 872
Schoeps, Johannes, 1024
Schomer, A., 720
Schroeder, C. Morrison, 331
Schulz, M. D., 164
Schumann, 361
Scott, J. V., 552
Scott, R. Kaye, 186
Scott, Roy W., 1039
Scott, Wendell G., 194, 372, 849
Scott, William H., 370
Seaman, William B., 849
Sear, H. R., 175
Seed, J. C., 365
Selverstone, B., 364
Sénèque, J., 506
Seybold, William F., 329
Shafer, Charles L., 364
Shallow, T. A., 1044
Shanks, S. Cochrane, 690
Shapiro, D. M., 365
Shapiro, David, 848
Sharpe, Myer, *769
Shaw, G. E., 879
Shaw, Robert R., 520
Sheinmel, A., 1033, 1034
Sherman, Alfred I., *75
Shifrin, Leo A., 171
Sherman, Mary S., 861
Shers, J. J., 849
Shimkin, Michael B., 367
Shipley, R. A., *576
Shipman, S. J., 523
Shull, J., 164
Shullenberger, W. A., 172
Sibley, J. A., 904
Sievert, R., 889
Sievert, Rolf M., 889
Siffert, Robert S., 347
Silverman, Frederic N., 547, *747
Simmonds, F. A., 176
Simon, M. S., 187
Simonetti, Marrigo, 688
Simril, Wayne A., 849
Singer, Jacob Jesse, 144
Sissons, H. A., 898
Skaggs, L. S., 722
Skanse, B., 362
Sklaroff, David M., 849, *968
Slater, M., *781
Slaughter, B. P., 358
Slavin, M., 701
Slavin, Paul, 524
Smalldon, Ted R., *375
Smelin, Ammiel, 711
Smets, Willy, 549
Smith, E. J., 890
Smith, H. V., 1025
Smith, L. A., 716
Smith, Marcus J., 166
Smith, Parke G., 847
Smith, R. R., 324
Smitham, J. Hillyer, 532
Sneedden, V. D., 324
Sniffen, Ronald C., 319
Sobel, Robert, 865
Soeur, R., 874
Solomkin, Mark, 1048
Sommers, Sheldon C., 339
Sosman, Merrill C., 30, 221, 529, 918
Sossai, Michele, 531
Southworth, John D., *624
Spain, David M., 679
Spalding, Charles K., 188
Speakman, T. J., 899
Spiegel, Milton B., *789
Spiers, F. W., 373
Spillman, Ramsay, 121
Spoont, Stanley I., 347
Spurling, R. Glen, 145
Spyckerelle, G., 332
Squires, A. H., 877
Starkloff, G. B., 873
Starr, Kenneth W., 856
Stauffer, H. M., 328, 687, 695, 849, 875
Stein, Joseph, 1034
Stein, Tobias, 720
Steinberg, Israel, *222, 686
Steiner, C. A., 165
Stephens, H. B., 523
Stephenson, Hack U., Jr., 1029
Stevens, Charles D., 905
Stevenson, V. L., 163, 166
Stewart, Paul H., 905
Stika, Edward A., 171
Stirling, W. Calhoun, 172
Stockler, A. L., 522
Stoll, Basil A., 355
Stone, R. S., 195, 574
Storaasli, J. P., *559, *576
Storch, C., *53, 545
Storer, Edward H., 900
Storrs, Richard P., 322
Stout, Arthur Purdy, 717
Stronge, R. Fawcett, 713
Sullivan, A. W., 173
Sullivan, Maurice, 368
Sussman, M. L., 686, *819
Sutton, David, 516
Svartz, Nanna, 549
Sweet, Richard H., 319
Sweet, W. H., 364
Swenson, Orvar, 336
Swindell, G. E., 181

Talbert, Pierre C., 1047
Talbott, J. H., 175
Tankin, L. H., 704
Tanner, C. H., 163
Tarlov, I. M., *974
Taylor, R. L., 851
Taylor, S., 1029
Teilum, Gunnar, 682
Terry, Luther L., 327
Theismann, Hans, 556
Thelander, H. E., 861
Theodos, P. A., 1032
Therkelsen, F., 345
Thomas, Albert G., 679
Thomason, I. S., 181
Thompson, J. J., 1027
Thompson, W. H., 328
Thorsen, Gunnar, 700
Thuilliez, A.-C., 368
Thumann, Robert C., Jr., *640
Thurn, P., 155
Tillisch, Jan H., 181
Tochilin, E., *475
Tod, Margaret, 892
Tod, Margaret C., 359
Tompkins, V. N., 1025
Toole, H., 158
Torgersen, Johan, 672, 682

Touroff, A. S. W., 325
Tovee, E. B., 904
Townsend, Ruth O., 713
Tracey, Martin L., 159
Tremaine, Myron J., 539
Tsevrenis, H., 368
Tucker, Beatrice E., 1048
Tucker, F. R., 893
Tucker, H. St. George, Jr., 674
Tuhy, John E., 324
Tullis, John L., 370
Turin, R. D., 545
Turletti Tola, A., 318
Turner, H. Midgley, 320

Ude, Walter H., 869
Ulfelder, Howard, 352
Ullmann, Henry J., 911
Ulrich, Robert P., *740
Updegrave, William J., 897

v. Albertini, A., 180
Vaas, A., 326
Valadares, Manuel, 372
van Buchem, F. S. P., 531
van der Werff, J. Th., 873
van Geertruyden, J., 551
Van Hazel, W., 322
Van Ordstrand, H. S., 876
Van Vermeskerken, J. L., 531
van Wien, A., 551
Van Winkle, Walton, Jr., 369
Vasconcelos, E., 551
Vest, M., 153
Vieten, Heinz, 528
Vogt, Alfred, 885, 1043
Vollmer, Hermann, 318

Waddell, William R., 319
Wagner, F. B., Jr., 1044
Wahlberg T., 889
Wainwright, William W., 187
Walker, A. Earl, 1027
Walker, J. E., 527
Waller, J. I., 172
Walther, O., 544
Ward, G. E., 887
Ware, P. F., 895
Warmoes, F., 543
Warren, Richard, 541
Warren, Shields, 879
Warres, H. L., 340
Warthin, Thomas A., 541
Watson, W. L., 324
Watson-Jones, Reginald, 343
Waugh, J. M., 336, 338
Weaver, F. M., 337
Weaver, James B., 174
Weber, Harry M., *929
Webster, J. E., 1023
Weinberg, S., 1027
Weinberg, T., 1028
Weise, H., 533
Weisel, Wilson, 681
Weiss, A. G., 323
Wellens, P., 332
Wells, Charles, 336, 705
Wells, Robert C., 682
Wener, J., 187
West, Harold D., *831
Whitcomb, Frances, 681
Whitener, Donald L., 1047
Whittenberger, J. W., 1041
Wiebe, Werner, 148
Wiener, L. R., 875
Wildervanck, L. S., 712
Wilhelmi, O. J., 170
Williams, Charles R., 374
Williams, Katherine, 721
Williams, S. F., 898
Willis, R. A., 177
Wilson, Hugh M., 328
Wilson, J. W., 848
Winkler, Harry, 864
Winsor, T., 1041
Wise, Robert E., *239, 675
Wissing, Egon G., 541
Withers, R. W., 547
Witz, J., 323
Wolf, B. S., *999
Wolf, Stewart, 159
Woltz, Blanche, 524
Wood, Ernest H., Jr., 666, 849
Wood, John L., 364
Wood, Paul, 1040
Woodruff, C. E., 526
Woodward, E. R., 900, 904
Woolner, L. B., 678
Wright, Claude-Starr, *907
Wright, W. S., 875
Wyatt, J. P., 849
Wylie, E. J., 530

Yannitelli, S. A., 526
Yearwood, H. M., 358
Young, Barton R., 313
Young, Cabell, Jr., 864

Zanes, R. P., 918
Zatzkin, Herbert, 522
Zeavin, I., 700
Zelman, Morris, 680
Zeman, Wolfgang, 883
Zettergren, Lennart, 691
Zimmer, E. A., 665
Zimmerman, Henry A., 1039
Zingaro, Angelo A., *785
Zinninger, M. M., 544

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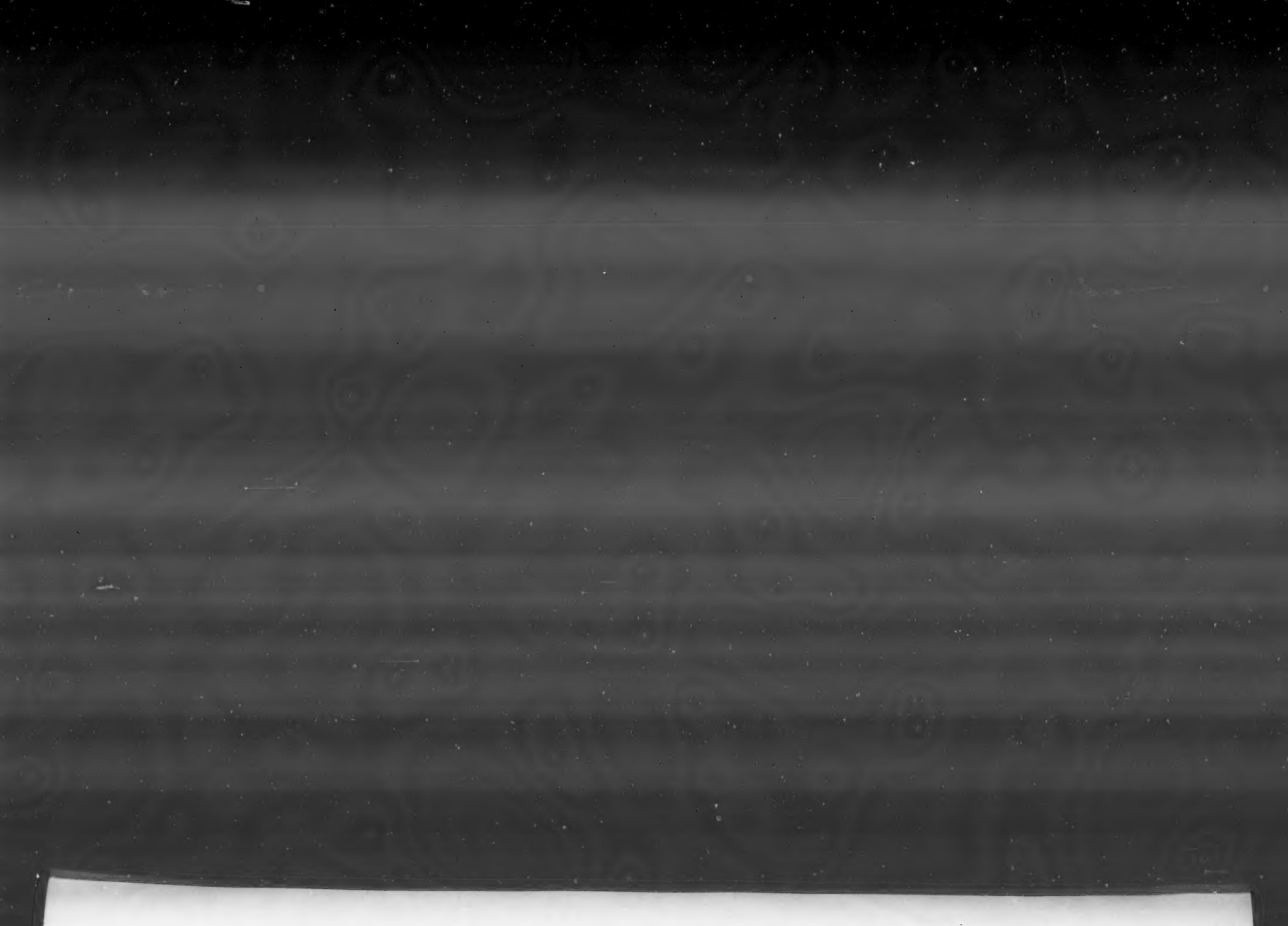
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